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INTRA-ABDOMINAL APOPLEXY

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This report will describe in detail only 1 case. However, since the pathologic process involved has been recorded in the literature only rarely, 12 previously reported cases will be tabulated and brief allusion made to them. In referring to intra-abdominal apoplexy, the subject of this report, Graham commented:

> This is probably a much more important matter than would seem to be the case from the small amount of discussion which it has received in the literature. Of interest also in this connection is the similarity of the symptoms produced by retroperitoneal hemorrhage to those of an acute surgical intra-abdominal lesion, a point which has been emphasized by Warren H. Cole.

Thompson and Dunphy stated:

> Intra-abdominal apoplexy is the spontaneous rupture of an arterio-sclerotic artery of one of the abdominal viscera. In this region it is the counterpart of cerebral hemorrhage, but, whereas the latter condition is common, intra-abdominal apoplexy is either extremely rare or seldom recognized.

As far as I have been able to determine, only 12 instances of this accident have been recorded in the literature. In 9 the condition was discovered at operation and in 3 at necropsy. In the thirteenth case, recorded here, it was discovered at operation.

REPORT OF A CASE

C. E. B., a white man aged 72, was admitted to the University of Virginia Hospital on March 24, 1936. He complained of severe, generalized cramplike pain in the abdomen, of approximately twelve hours' duration. The abdominal discomfort was so persistent and severe that the patient was referred for surgical consultation. The symptoms first appeared when he awakened in the morning; because he had not had a stool in two days, he attributed the pain to constipation and took a saline laxative. The constipation was not relieved until he received an enema, but even this did not alleviate the pain. He did not have nausea but induced vomiting, which did not relieve him.

Nearly two weeks previously, on March 13, herpes had appeared on his forehead. He had been treated by his physician on three successive days, March 18, 19 and 20, with 1 cc. of solution of posterior pituitary administered subcutaneously.

The herpes had improved promptly, and he had not had any untoward effects or symptoms in connection with the treatment. His history was otherwise unimportant.

The data obtained from examination did not suggest that the patient was very ill. He complained of pain in his abdomen, but it did not seem to be severe. Except in the abdomen, little of an abnormal nature was observed. The rectal temperature was 99.8 F.; the pulse and respiratory rates, normal. The blood pressure was not recorded. The abdomen was slightly distended and a little tender throughout, though there was no spasm of the muscles. No masses were palpable, and no signs of fluid were present. Examinations in the laboratory did not reveal any abnormalities. The hemoglobin content was 80 per cent (Dare), the erythrocyte count 4,020,000, the leukocyte count 8,200 and the differential count normal. The urine contained no abnormal constituents, and the Wassermann and Kahn reactions of the blood were negative.

Instances of Intra-Abdominal Apoplexy Recorded in the Literature

			Data					
No.	Year	Author	Sex	Age	Anatomic Location	Operation	Result	Autopsy
1	1913	Florence and Ducuing..	F	..	Sup. mesent. art.	No	Died	Yes
2	1918	Hilliard.....	M	48	Transv. mesocolon	Yes	Died	No
3	1923	Starcke.....	M	60	Gast.-duod. art.	Yes	Recovered	No
4	1925	Budde.....	M	27	L. gast.-epip. art.	Yes	Recovered	No
5	1931	Green and Powers.....	F	54	L. gast. art.	Yes	Recovered	No
6	1933	Mourgue - Molines and Cabanac	M	56	L. gast. art.	Yes	Recovered	No
7	1933	Mourgue - Molines and Cabanac	..	..	L. gast. art.	Yes	Recovered	No
8	1933	Mourgue - Molines and Cabanac (Rud)	F	73	L. gast.-epip. art.	Yes	Died	Yes
9	1935	Thompson and Dunphy	F	62	L. gast. art.	Yes	Recovered	No
10	1935	Buchbinder and Greene	M	57	R. gast. art.	Yes	Recovered	No
11	1936	Moorehead and McLester	M	44	R. and l. gast. art.	No	Died	Yes
12	1936	Moorehead and McLester	M	50	Sup. mesent. art.	No	Died	Yes
13	1938	Morton	M	72	Sup. mesent. art.	Yes	Recovered	No

During two days in the hospital, the patient's symptoms gradually subsided, and the distention and tenderness in the abdomen disappeared almost entirely. The tenderness, however, persisted to a slight degree, in the lower part of the abdomen on the right side more than elsewhere. It was therefore thought that the patient might have had a mild attack of appendicitis, although a positive diagnosis was not made at that time. Accordingly, when he was discharged, he was advised to restrict his diet somewhat, to avoid the use of laxatives and to report immediately if the symptoms reappeared.

Nothing further was heard from him until five days later, March 30. His wife telephoned early in the morning to relate that he had been awakened by severe cramps in the abdomen. He was at once readmitted to the hospital. Before coming to the hospital, however, he had taken an enema that had not been effectual or relieved the pain. He had been nauseated and had vomited. The results of examination at the time of readmission were essentially the same as those at the time of the former admission. There was, however, a little more tenderness throughout the abdomen, and in addition there was an indefinite mass to the left of the umbilicus. The patient's temperature was 100 F., and his pulse and respiratory rates were only slightly elevated. It was thought that he probably had an abscess in the left side of the abdomen, possibly the result of diverticulitis

of the colon. Further observation seemed more desirable than immediate operation. During the day the patient gradually became more comfortable, and the tenderness in the abdomen decreased perceptibly. He spent a reasonably comfortable night, but at 6 a. m. he was seized again with severe cramping pain in the abdomen. About three hours later, rather suddenly, he went into a state of shock. After appropriate measures to combat shock had been employed successfully, plans were made for an immediate exploratroy laparotomy.

Operation was performed on March 31. With the patient under anesthesia induced by inhalation of ether, the abdomen was explored through an incision in the middle portion of the rectus muscle on the left side. The incision overlay the mass which had been palpated just to the left of the umbilicus. Immediately on incision of the peritoneum, a large amount of blood escaped. Investigation revealed the presence of much liquid blood as well as a quantity of blood clot throughout the abdominal cavity. It was estimated that there must have been at least as much as 500 cc. of each. There was a hematoma in the root of the mesentery of the small intestine, which was approximately 10 cm. in diameter The inferior aspect of the hematoma had ruptured and permitted the extravasation of the blood observed in the free peritoneal cavity. The source of the hematoma was obviously a rupture of one of the branches of the superior mesenteric artery, in or near the root of the mesentery. A more exact identification of the vessel was precluded by the nature and location of the lesion. It seemed unwise to tamper with the hematoma and take the chance of starting a fresh hemorrhage or endangering the blood supply to the intestine. Because there was no evidence of active bleeding after the blood and clots had been removed from the abdomen, the incision was closed without any further attempt at operative procedure. The usual suture of the abdominal wall in layers was carried out in the routine manner, and the patient was returned to his room in excellent condition, apparently none the worse for the operation.

Postoperative convalescence was somewhat prolonged by slow healing of the incision, but uneventful. Several transfusions of blood were given at intervals in order to restore the blood lost by hemorrhage. Repeated determinations of blood pressure did not reveal any arterial hypertension. The patient was discharged from the hospital on May 11, forty-one days after the operation. At the time of discharge, he was able to walk about and his general condition seemed to be good. Follow-up reports have been interesting in that the patient apparently has been in as good health since the operative demonstration of intra-abdominal apoplexy as before. He was a patient in the hospital in the medical service for acute infection of the respiratory tract from Dec. 14, 1936, to Jan. 5, 1937. At the time of that admission his blood pressure was 155 systolic and 96 diastolic. At the time of discharge the blood pressure was 100 systolic and 70 diastolic. At the time of writing, more than one year after the occurrence of the hemorrhage, the patient is in good health. He has no complaints referable to the original disease except a slight weakness of the abdominal wall at the site of the operative incision.

COMMENT

Reference to the accompanying table and comparison of this case with those previously reported by others do not reveal any striking differences except in the location of the lesion. This is only the third instance in which the hemorrhage has occurred in a branch of the superior mesenteric artery. The age of this man was above the average

in the cases previously reported but not the highest on record, for the patient in 1 case was a year older. As for incidence in the sexes, occurrence in men seems to predominate over that in women by a ratio of 2 to 1. As in all except 3 of the other cases, the diagnosis was made in the course of operation. It is interesting that the correct diagnosis has not been made before operation in a single instance. In the 3 cases in which an operation was not performed, the diagnosis was not made prior to necropsy. In each case in which an operation was performed, the preoperative diagnosis was that of some acute abdominal emergency, such as perforated viscus, acute inflammatory process or acute intestinal obstruction. Of the conditions mentioned as tentative preoperative diagnoses, that most nearly related to the true condition was mesenteric thrombosis. Probably the most interesting fact about abdominal apoplexy is that, in spite of its similarity to cerebral apoplexy, a condition frequently encountered, it has rarely been observed. Of course, it may occur much more frequently than published reports would indicate; nevertheless, even though allowance is made for this discrepancy, the condition must still be rare in comparison with the incidence which might be expected.

The reported cases were almost all those of persons in the period of life in which arteriosclerosis is always present to some degree. In cases in which tissue was examined there was both gross and microscopic evidence of arteriosclerosis. There would seem to be no doubt, therefore, that arteriosclerosis is the underlying pathologic process usually responsible for the rupture of the vessel and the consequent hemorrhage. The precipitating cause of the rupture may be, as in the case of cerebral hemorrhage, unexplainable. Anything that raises blood pressure might well predispose to a blowing out at any previously weakened spot. In the case here reported a pressor drug had been administered hypodermically a few days before the first abdominal symptoms appeared. It seems fair to assume that this drug may have played a part in the rupture of the vessel. On the other hand, it is not uncommon for cerebral hemorrhage to occur while one is asleep, although blood pressure is then presumably at its lowest and most stable level. If one may reason by analogy, one may conclude that there need not be any precipitating cause for the hemorrhage. The vessel wall in a diseased area merely becomes gradually too weak to withstand even normal systolic pressure and finally ruptures. All these considerations still leave unexplained the rarity of intra-abdominal apoplexy.

Spontaneous rupture of an aneurysm within the abdominal cavity is an accident that is pathologically and clinically related to intra-abdominal apoplexy. It occurs much more frequently, however, perhaps because of the difference in effective pressures in the larger and the smaller vessel, and is usually immediately fatal. Instances of rupture of aneurysm of the aorta are not uncommon, and the same is true to a

lesser degree in the splenic and other large arteries within the abdominal cavity. It is possible, of course, that in some of those cases recorded as instances of true intra-abdominal apoplexy the rupture of the vessel may have occurred in a small preexisting aneurysm. If this was the case, however, the pathologic proof of it was overlooked.

The diagnostic problem presented by cases of intra-abdominal apoplexy seems to be almost insurmountable. A correct clinical diagnosis was not made in a single case of all those so far reported in the literature, and that reported here is no exception. An examination of the pathologic process involved and the clinical symptoms does not disclose any characteristic that is sufficiently distinctive to offer much hope for the recognition of cases that may be encountered in the future. It would seem that the condition must be included in the group of acute diseases that warrant surgical exploration of the abdomen because of signs of severe irritation of the peritoneum. If the condition is borne in mind as a possibility when one is dealing with diseases, it is quite possible that it may be occasionally diagnosed, at least presumptively.

Treatment, if one may judge by the reported cases, must usually be surgical. All except 3 of the patients were subjected to operation, and those 3 died rather suddenly of massive hemorrhage. Of those operated on, only 2 of 10 died, 1 at the end of six hours and the other after six days. The operative procedure employed was ligation of the bleeding vessel in 4 instances, while in the other 6 apparently nothing was done. In the case here reported nothing was done because there was no evidence of active bleeding. Moreover, it was feared that to disturb the hematoma at the root of the mesentery of the small intestine would endanger the circulation in the intestine. It seems reasonable to suppose, however, that ligation of the bleeding vessel may in some instances be the means of preventing continued loss of blood and consequent death. Immediate postoperative treatment and later after-care of the patient would not seem to offer any particular problems. General measures to promote a serene life should be advised, just as in the case of cerebral hemorrhage. Pressor drugs should no doubt be avoided. In this connection it is interesting to note that no case of a second attack of intra-abdominal apoplexy has ever been reported, although instances of repeated cerebral apoplexy are common.

SUMMARY

Twelve cases of intra-abdominal apoplexy, the spontaneous rupture of an arteriosclerotic artery of one of the abdominal viscera, were found in the literature and are briefly discussed. In connection with them, an additional case, which I encountered, is reported in greater detail. The etiology of the disease, as far as the pathologic process in concerned, seems plain, though its rarity and the factors that may precipitate it

are unexplained. The condition does not seem to have any distinctive characteristics that may lead to a positive diagnosis. It should, however, be thought of as a possibility in any case in which there are signs of peritoneal irritation. Treatment is surgical and should be by ligation of the bleeding vessel whenever practicable.

BIBLIOGRAPHY

Buchbinder, J. R., and Greene, E. I.: Intra-Abdominal Apoplexy, J. A. M. A. **105**:874-875 (Sept. 14) 1935.

Budde, M.: Operativ geheilte Spontanruptur der Arteria gastro-epiploica sinistra, München. med. Wchnschr. **72**:1383-1384 (Aug. 14) 1925.

Florence, M., and Ducuing, M.: Contusion du rein; hémopéritoine; guérison spontanée; valeur diagnostique de la ponction exploratrice du cul-de-sac de Douglas, Bull. et mém. Soc. nat. de chir. **39**:645-650, 1913.

Graham, E. A.: The 1936 Year Book of General Surgery, Chicago, The Year Book Publishers, Inc., 1936, p. 276.

Green, W. T., and Powers, J. H.: Intra-Abdominal Apoplexy, Ann. Surg. **93**: 1070-1074 (May) 1931.

Hilliard, J. W.: Spontaneous Hemorrhage into the Peritoneal Cavity in Arteriosclerosis, Brit. M. J. **1**:231 (Feb. 23) 1918.

Moorehead, M. T., and McLester, J. S.: Abdominal Apoplexy: Fatal Intra-Peritoneal Hemorrhage Due to Spontaneous Rupture of a Visceral Artery, J. A. M. A. **106**:373-374 (Feb. 1) 1936.

Mourgue-Molines, E., and Cabanac: Abondante hémorragie intrapéritonéale par infarctus de l'épiploon-gastro-hépatique, Bull. et mém. Soc. nat. de chir. **59**:720-727 (May 13) 1933.

Rud, cited by Mourgue-Molines, p. 725.

Starcke, G.: Spontaneous Rupture of Gastroduodenal Artery, Ugesk. f. læger **85**:963-964 (Dec. 27) 1923.

Thompson, K. W., and Dunphy, J. E.: Intra-Abdominal Apoplexy, Ann. Surg. **102**:1116-1118 (Dec.) 1935.

ACUTE RETROCECAL APPENDICITIS

BASED ON SEVEN HUNDRED AND FIFTY-ONE INSTANCES

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LOS ANGELES

In view of the fact that during the past 10 years nearly four thousand articles have crowded the medical literature on the general subject of appendicitis, it is indeed strange that its most dangerous form, namely, acute retrocecal appendicitis, has been so largely neglected. The classic article by Brunn on acute pelvic appendicitis has done much to direct attention to the relation existing between the position of the

TABLE 1.—*Average Distribution in Percentage of Diseased Appendixes According to the Pathologic Condition Encountered*

Author and Year	Number of Cases Reported	Acute Appendicitis with Unruptured Appendix	Acute Appendicitis with Perforated Appendix and Abscess or Localized Peritonitis	Acute Appendicitis with Spreading or Generalized Peritonitis
Various authors,* 1931 to 1937.....	30,593	53.8	30.8	19.4
Collins, 1938.....	751	47.35	31.12	21.42

* Bower; Finney; Keyes; Wevill and Wallace; Harris; Cayford; Lied; Merrell; Reid, Poer and Merrell; Lamon; Hobler; Pattison; Cook and Beardsley; Rhodes, Birnbaum and Brown, and Sperling and Myrick.

appendix and acute inflammation. Acute retrocecal appendicitis is largely responsible for the atypical signs and symptoms in cases of acute appendicitis that deceive the incautious diagnostician and cause many deaths. In my previous studies it has been proved that most adherent retrocecal appendixes derive their abnormal position from previous attacks of acute appendicitis. Wakeley studied the position of the human vermiform appendix in 10,000 cases. He reported that in 65.28 per cent of his series the appendix was either free or adherent in a postcecal or retrocecal position. In a study published in 1932, on postmortem examinations of 4,680 appendixes, I noted that only 20.21 per cent were fixed in a retrocecal situation. Meyer and Spivack studied 370 cadavers and observed that 9.5 per cent of the appendixes were true retrocecal specimens.

During the past 10 years, I have encountered 751 instances (25.0 per cent) of acute retrocecal appendicitis among 3,003 consecutive

appendectomies performed for acute appendicitis; the diagnosis was verified by the pathologist in each case. Study of the 751 histories emphasizes the frequently bizarre nature of the early signs and symptoms presented by patients with acute retrocecal appendicitis.

Four hundred and seventy-one (62.72 per cent) of the patients were men. Krasnoselskiy stated the accepted opinion that the incidence of acute appendicitis is less in women, that the condition runs a milder clinical course, and that the mortality is about one third of that occurring in men. Table 2 supports this contention. The average age of the 751

TABLE 2.—*Mortality and Distribution of Diseased Appendixes According to Sex*

			Males				Females			
			Cases		Deaths		Cases		Deaths	
Author	Year	Number of Cases	Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage
Finney	1933	1,807	1,111	61.5	65	5.85	696	38.5	24	3.45
Wevill and Wallace	1934	8,265	4,652	56.2	241	5.2	3,613	43.8	129	3.6
Lamon	1936	206	.....	....	8	....	.....	....	6	....
Boyce and others	1936	2,715	1,848	68.0	106	5.7	867	32.0	51	5.9
Collins	1938	751	471	62.72	41	8.7	280	37.28	17	6.07

TABLE 3.—*Distribution of Diseased Appendixes by Age Group, Expressed in Percentage*

		No. of	Age Groups by Years								
Author	Year	Cases	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	80+
Pattison	1936	1,211	10.47	38.3	29.1	10.0	5.78	3.55	1.73	0.33	0.08
Reid and others	1936	3,100	6.6	28.3	36.4	18.7	5.78	2.81	1.1	0.29	0.00
Pool	1936	757	10.7	41.3	27.7	13.4	4.35	2.11	0.26	0.00	0.00
Rhodes and others	1936	1,000	8.3	32.7	30.7	13.4	8.3	3.6	1.9	0.3	0.2
		6,068									
Average percentage			9.02	35.15	30.97	13.9	6.05	3.02	1.25	0.31	0.14
Collins	1938	751	6.25	15.16	40.43	21.55	9.44	4.25	2.13	0.66	0.00

persons was 29.7 years. This age is higher than that usually reported for acute appendicitis. Three hundred and four instances (40.48 per cent) occurred in the third decade of life. There is a noticeable increase in the incidence of this type of appendicitis in the later decades, as depicted in table 3.

Racial and seasonal incidence showed no noteworthy features. The various occupations and previous residences of these persons were unimportant in respect to this study. Interestingly, 267 persons (35.55 per cent) presented histories of other members of their families having suffered attacks of appendicitis that necessitated surgical intervention.

The past histories of these persons were of great importance. Three hundred and one (40.08 per cent) mentioned the previous frequent occurrence of infections of the upper part of the respiratory tract, and in

111 of these (14.78 per cent) such infections had preceded their present acute abdominal complaints by an average interval of 8.4 days. One hundred and thirty-six patients (18.11 per cent) had had recurring attacks of chronic sinusitis of one type or another. Three hundred and seventeen persons (42.21 per cent) still possessed their tonsils or adenoids, and for 293 (38.88 per cent) diagnoses of chronic tonsillitis were recorded in the histories. The most interesting fact in these histories was that 466 persons (62.05 per cent) had had previous similar attacks of abdominal pain of varying degrees of severity. (See table 4 for further details.) One hundred and fifty-one persons (20.11 per cent) had been told in the past by their physicians that they had appendicitis which required immediate surgical intervention. For one reason or another, on receiving this advice, they had failed to permit the performance of surgical abdominal explorations. Of those who had had previous similar attacks of abdominal pain, 113 (15.05 per cent)

TABLE 4.—*Percentage of Patients Having Had Previous Attacks of Appendicitis*

Author	Year	Number of Cases	Previous Attacks	
			Number	Percentage
Merrell	1935	2,921	1,200	41.12
Klein	1935	86	23	26.7
Reid and others	1936	2,035	837	41.12
Pool	1936	757	222	27.1
Boyce and others	1936	2,715	713	26.3
		8,514		
Average	....	.....	599	35.2
Collins	1938	751	466	62.05

had experienced more than one attack, the average number being 2.4. In the 151 cases, 119 appendixes (15.85 per cent) were gangrenous, 74 (9.85 per cent) had perforated and 13 patients (1.74 per cent) died.

One hundred and thirty-eight persons (18.39 per cent) had been previously treated for cholecystitis of varying severity. Ninety-nine others (13.18 per cent) had been considered in the past as suffering from some form of gastric or duodenal disease. Seventy-one additional patients (9.45 per cent) had received treatment for disease of either the right kidney or the right ureter. Of course, it must be admitted that in all probability some of these persons did actually have such diseases independent of their subsequent attacks of acute retrocecal appendicitis. Sixty-four persons (8.52 per cent) had had cholecystectomies for chronic cholecystitis without cholelithiasis, but had not been relieved of their chief complaints. Fifty-two persons (6.79 per cent) had been operated on for various types of peptic ulcer, without alleviation of their symptoms.

Of the 280 women of this series, 44 (5.86 per cent) had received medical treatment for pelvic inflammatory disease, without obtaining noticeable relief. At the time of the appendectomy, 33 (4.394 per cent) showed no evidence of the previous existence of any pelvic inflammatory disease. Two (0.27 per cent) nephropexies had been performed in the past for conditions in the right side of the abdomen, without relieving the patients. Forty-five women (5.99 per cent) had been subjected to various gynecologic procedures, without experiencing permanent relief. An additional 31 (4.13 per cent) had had major pelvic operations of various types, at which, for one reason or another, their appendixes had not been removed.

There were thus 147 persons (19.57 per cent) whose abdomens had been opened previously for other suspected intra-abdominal diseases, but during these operations their appendixes had not been removed, and they subsequently suffered attacks of acute appendicitis. Therefore, most of these persons had been subjected to needless secondary operations, at a time when the operative risk and the postoperative morbidity were greatly increased. Most regrettable was the fact that 6 (0.80 per cent) died. Of the 141 patients (18.77 per cent) with histories of previous abdominal explorations who survived, 131 (92.91 per cent) were relieved of their persistent complaints. Thus, 56.99 per cent of this entire series of 751 patients had received treatment in the past for diseases of the gallbladder, the pyloric region, the pelvis or the right part of the genitourinary tract. Gynecologic operations were the vogue among the 280 women of this series, while operations on the upper part of the abdomen were fashionable among the 471 men.

Nausea, headache, flatulence, heartburn, regurgitation, insomnia, constipation and lack of appetite, of which many of these patients complained, disappeared in 73.4 per cent of the cases after appendectomies. Distress following the ingestion of cheese, rich or greasy fried foods and raw fruits likewise frequently vanished after the removal of the diseased appendixes. Many persons who complained of symptoms simulating either cholecystitis or peptic ulcer were either cured or greatly relieved after appendectomies. In 12 (1.60 per cent) persons over 55, certain long-continued arthritic pains disappeared after the removal of their appendixes. In 21 persons (2.80 per cent) of the same age group, similar symptoms were greatly diminished by appendectomies, which appreciably increased their usefulness both to themselves and to others.

One hundred and thirty-seven patients (18.24 per cent) complained of mild pains of several days' duration in the epigastric or the right hypochondriac region, which failed to become localized at McBurney's (or more properly, McArthur's) point in the right lower quadrant of the abdomen. Two hundred and fourteen persons (28.50 per cent)

complained of steady, dull, deeply situated pains in the right lumbar region of the back. Eighty-eight persons (11.72 per cent) experienced pains in the right shoulder. In 107 instances (14.25 per cent) severe pains in the upper part of the abdomen continued for an average of 6.4 hours. This syndrome was followed by a cessation of the pains and a temporary sense of well-being. However, after the elapse of an average of 4.9 hours, this period of calm was replaced by nausea, vomiting, a feeling of grave illness and diffuse, colicky pains. Only 142 persons (18.91 per cent) presented the fairly classic signs and symptoms of acute appendicitis described in textbooks, and only 2 (0.27 per cent) of this subgroup died of generalized peritonitis.

The average temperature in the mouth at admission was 100.4 F. The pulse rate averaged 88 beats per minute. The average number of respirations was 18 per minute. The average blood pressure per age group, when compared to published normal values, was 30 mm. of mercury less for the systolic and 18 mm. of mercury less for the diastolic reading. The facies and the general appearance of the patients did not usually suggest, on first glance, that they were really sick. One hundred and four persons (13.85 per cent) revealed the existence of either recent or subsiding acute infections of the upper part of the respiratory tract, when the nasopharynx and the lungs were examined. Four hundred and nineteen instances (55.79 per cent) were recorded of the existence of advanced degrees of dental caries or pyorrhea alveolaris. In 57 histories (7.59 per cent), it was recorded that the right side of the diaphragm was limited in its excursion.

The physical examination showed little of interest until the abdomen, back and rectum or pelvis were examined. Inspection of the abdomen usually revealed only the presence of distention in its upper part. Palpation presented a diffuse, mild, generalized tenderness in the right side of the abdomen located lateral and cephalad to McArthur's point. Hyperesthesia of the skin was rarely seen in the right lower quadrant of the abdomen. Muscular rigidity was appreciably less than is usually met with in cases of acute appendicitis and was absent in 397 instances (52.86 per cent). Rebound tenderness was not easily elicited and was absent in 243 cases (32.36 per cent).

The sign most frequently obtained was the production of intense pain by exerting steady, deep pressure on the right flank, lateral to the right psoas major muscle. Percussion was of little value, while auscultation of the abdomen usually gave no remarkable results. In 375 patients (49.93 per cent) the rectal examination gave no sign of tenderness or of a mass in the right side of the pelvis. Yet the rectal examination in 218 instances (29.03 per cent) gave the only positive signs of the existence of acute appendicitis.

The average leukocyte count in this series was 11,870 cells per cubic millimeter of whole blood, with an average differential count of 83.0 per cent of polymorphonuclear neutrophils. Thus, the blood showed a slighter leukocytosis than is usually present in cases of acute appendicitis. Interestingly, the urine of 104 patients (13.85 per cent) contained an appreciable number of erythrocytes, while pyuria was recorded in 152 histories (20.24 per cent), evidence being present in even the catheterized samples. Such urinary observations often were misleading and usually caused operative delay until diagnostic procedures could be utilized to rule out the existence of disease in the right part of the genitourinary system. These patients when operated on later frequently revealed an acutely inflamed appendix lying across the course of the right ureter, with an accompanying secondary acute periureteritis. For 37 women (4.93 per cent) sedimentation rates were determined in an effort to rule out the possible existence of pelvic inflammatory disease. In only 3 instances (0.4 per cent) were the rates abnormal. These observations thus agree with those previously made by Lesser and Goldberger that the sedimentation rate for women suffering from acute appendicitis is normal. (See table 5 for further details.)

Correct preoperative diagnoses of acute appendicitis were made in only 554 cases (73.77 per cent). In the recent study by Rhodes, 75 of his preoperative diagnoses were tabulated as follows: correctness on the question of perforation in 58.6 per cent of the instances, on the question of the location of the appendix in 40.0 per cent and on the question of local pathologic changes in 33.3 per cent. In 136 persons (18.11 per cent) erroneous preoperative diagnoses were made of acute perforated peptic ulcer. In 53 additional persons (7.06 per cent) preoperative diagnoses were made of acute empyema of the gallbladder with impending perforation. Five persons (0.67 per cent) were considered to have acute pancreatitis. Three patients (0.40 per cent) were decided to be suffering from acute mesenteric vascular occlusions.

Of the 554 cases (73.77 per cent) in which correct preoperative diagnoses of acute appendicitis were made, only 304 (40.48 per cent) were correctly diagnosed as of the retrocecal variety. In these 304 cases, as a result of such preoperative diagnoses, 277 (36.88 per cent) lateral and cephalad placed muscle-splitting incisions (rather than the usual McBurney incisions) were made in the right lateral anterior abdominal wall. This type of incision gave an excellent exposure of the retrocecal region and permitted the performance of appendectomy under direct vision, the latter point being a most important factor in lessening postoperative morbidity. (See table 6 for further information.) No herniations occurred postoperatively with this type of muscle-splitting incision. The period of hospitalization of these patients was on

TABLE 5.—*Important Signs and Symptoms Presented in This and Other Series of Cases of Appendicitis*

Signs and Symptoms	Kline, 1935	Reid and Others, 1936	Boyce and Others, 1936	Finney, 1933	Collins, 1938
Nausea	51.0%	78.0%	.....	.....	56.66%
No nausea	.....	.....	2.14%	.....	.....
Vomiting	45.0%	73.0%	.....	.....	53.33%
No vomiting	.....	.....	2.14%	.....	.....
Constipation	31.0%	.....	.....	.....	28.46%
Diarrhea	.....	.....	1.71%	.....	13.43%
Pain in right shoulder	.....	.....	.....	.....	11.72%
Fever	50.0%	.....	.....	.....	68.89%
High fever (102-105 F.)	.....	.....	4.8%	.....	11.17%
Normal temperature	32.0%	.....	20.8%	.....	18.75%
Subnormal temperature	16.0%	.....	.....	.....	12.36%
Leukocytosis (over 10,000 cells)	46.0%	.....	.....	.....	69.43%
White blood count below 10,000	.....	.....	17.2%	.....	30.56%
Average leukocyte count	.....	17,200	.....	17,669	11,870
Average polymorphonuclear count	.....	.....	.....	85.5%	83.0%
Polymorphonuclear count over 80%	41.0%	.....	.....	.....	58.17%
Erythrocytes in urine	9.0%	.....	.....	.....	13.85%
Albuminuria	28.0%	.....	.....	.....	23.71%
Casts in urine	12.0%	.....	.....	.....	10.11%
Pyuria in catheterized urine specimen	.....	.....	.....	.....	20.24%
Localized pain in right lower quadrant of abdomen	81.0%	.....	.....	.....	18.88%
Pain elicited in right flank	.....	.....	.....	.....	63.31%
Muscular spasm localized in right lower quadrant of abdomen	52.0%	.....	.....	.....	47.20%
Limited excursion of right side of diaphragm	.....	.....	.....	.....	7.50%
Boardlike muscular rigidity	10.0%	.....	.....	.....	4.52%
Sedimentation rate abnormal	.....	.....	.....	.....	8.11%
Use of Penrose drains	42.0%	.....	.....	.....	34.84%
Rebound abdominal tenderness	35.0%	.....	.....	.....	67.64%
Abdominal pain	.....	94.0%	96.3%	.....	83.12%
Rectal tenderness	.....	44.0%	.....	.....	50.07%
Typical history of appendicitis	.....	53.0%	.....	.....	18.91%
Average temperature	.....	100.1 F.	.....	.....	100.4 F.
Average pulse rate	.....	99	.....	.....	88
Typical history of pelvic disease	.....	.....	0.50%	.....	6.62%
Typical history of perforated peptic ulcer	.....	.....	.....	.....	18.11%
Typical history of cholecystitis	.....	.....	0.44%	.....	7.00%
Typical history of renal disease	.....	.....	0.07%	.....	11.17%
Typical history of ectopic pregnancy	.....	.....	0.07%	.....	0.00%
Association with respiratory disease	.....	.....	7.59%	.....	13.85%
Association with alcoholism	.....	.....	1.43%	.....	9.57%
Association with dietary indiscretions	.....	.....	3.08%	.....	16.49%
Pain in left side of abdomen only	.....	.....	0.79%	.....	1.19%
No localization of pain in right lower quadrant of abdomen	.....	.....	2.23%	.....	18.24%
Advanced dental caries, pyorrhea	.....	.....	.....	.....	55.79%

TABLE 6.—*Incidence of Postoperative Complications in Reference to Type of Incision Used*

	Type of Incision			
	Muscle-Splitting	Midright Rectus	Midline	Totals
Number of cases	277	313	161	751
Postoperative complications				
Postoperative morbidity	77 (10.25%)	178 (23.70%)	141 (18.73%)	396 (52.73%)
Pelvic abscesses	24 (3.20%)	31 (4.13%)	42 (5.50%)	97 (12.92%)
Secondary closures of wound	0 (0.00%)	10 (1.33%)	21 (2.80%)	31 (4.13%)
Hernias after incision	0 (0.00%)	17 (2.20%)	34 (4.53%)	51 (6.79%)
Average number of days in the hospital	16	23	27	
Average time spent at performing operation	37 min.	51 min.	62 min.	
Sex: Males	251 (33.42%)	172 (22.90%)	48 (6.39%)	471 (62.72%)
Females	26 (3.46%)	99 (13.18%)	155 (20.64%)	280 (37.28%)
All deaths	12 (1.60%)	18 (2.40%)	28 (3.73%)	58 (7.72%)

an average 9 days shorter and only one-eighth as many postoperative complications occurred among them as among those having had either midright rectus or midline incisions. Consult table 6.

However, in instances in which a doubt existed as to the cause of the acute condition in the abdomen, preliminary exploratory midright rectus incisions were made at the level of the umbilicus, so that they could be extended either caudally or cephalically as the intra-abdominal observations dictated. In 77 patients (10.25 per cent) walled-off retrocecal abscesses were palpated through such exploratory incisions. These

TABLE 7.—*Tabulation of Postoperative Complications in This and Other Series of Cases of Acute Appendicitis*

Postoperative Complications	Author and Number of Cases Reported				
	Boland, 4,270	Wevill and Wallace, 8,265	Pattison, 1,148	Sperling and Myrick, 433	Collins, 751
Ileus	33	..	..	..	23
Fecal fistula	7	59	..	..	2
Enterostomy	18	50	9	..	6
Bronchitis	..	150	..	..	59
Bronchopneumonia	12	19	7	..	56
Lobar pneumonia	0	46	..	..	10
Pulmonary atelectasis	..	13	..	..	19
Pulmonary embolism	2	8	..	..	0
Empyema	1	8	..	..	4
Cardiac disease	3	..	..	..	19
Peritonitis	335	1,274	129	68	161
Pleurisy	..	18	..	..	22
Lung abscess	..	1	..	..	6
Infected incision	..	234	67	22	98
Incomplete intestinal obstruction	..	56	..	..	14
Acute intestinal obstruction	..	67	9	5	22
Peritonitis with obstruction	..	22	..	..	9
Subdiaphragmatic abscess	..	17	2	..	11
Subhepatic abscess	..	..	2	..	7
Thrombosis and phlebitis	..	32	..	..	26
Pelvic abscess	..	30	41	..	97
Intra-abdominal abscess	..	23	11	..	31
Appendical abscess	197	1,123	268	107	234

were then closed without disturbing the abscesses and secondary muscle-splitting incisions were made directly over the abscesses and proper drainage instituted.

In the remaining 474 cases (63.12 per cent), in which either midright rectus incisions (313—[41.68 per cent]) or midline incisions (161—[21.44 per cent]) were employed, the appendixes were removed usually through lengthened incisions with considerable difficulty. This necessitated increased retraction of the edges of the wound, with a resultant increased degree of trauma to the anterior abdominal wall. Thirty-one (4.12 per cent) such incisions required a secondary closure before the patients could be dismissed from the hospital. In 51 additional cases (6.79 per cent) hernias of varying degree developed after incision. There were 319 examples (42.48 per cent) of troublesome complications

that developed in the non-muscle-splitting incisions. (Consult table 6 for further details.) The average stay in the hospital for patients with incisions of this type was 25 days, as compared to 16 days for those with muscle-splitting incisions.

Localized abscesses were drained by the use of simple Penrose drains of soft rubber dam material. These drains were completely removed

TABLE 8.—*Average Number of Days Spent in the Hospital According to the Pathologic Condition Found*

Author	Year	Number of Cases	Simple Acute Appendicitis	Appendicitis with Abscess	Appendicitis with Generalized Peritonitis
Finney	1903	3,913	14.3	28.8	34.7
Merrell	1905	2,921	8.8	....	27.3
Reid and others	1936	2,921	8.21	....	27.3
Sperling and Myrick	1937	433	9.75	22.0	23.0
Collins	1938	751	16.0	21.5	26.0

TABLE 9.—*Mortality in Relation to Type of Appendicitis*

Type of Appendicitis	Number of Cases	Percentage of 751	Number of Deaths	Percentage of 751	Percentage of Type	Mortality Due to Appendicitis
Acute simple appendicitis	154	20.50	1	0.133	0.65	0.000%
Nonperforated gangrenous appendicitis	202	26.90	10	1.332	4.95	1.064%
Appendicitis with abscess or localized peritonitis	234	31.16	15	1.997	6.41	1.862%
Acute appendicitis with perforated appendix and general peritonitis	161	21.44	32	4.261	19.88	3.481%
Totals	751	100.00	58	7.723		6.407%

TABLE 10.—*Causes of Death in This Series*

Condition	Number of Cases	Number of Deaths	Percentage of 751	Percentage of Condition
Pelvic abscess	97	12	1.60	12.37
Subdiaphragmatic abscess	11	4	0.53	36.36
Liver abscess and ascending pylephlebitis	8	8	1.06	100.00
Pneumonia	68	9	1.20	13.24
Generalized peritonitis	113	25	3.33	22.12
Totals	297	58	7.72	

by the twelfth day after operation. Drainage was not instituted in instances of generalized peritonitis, the peritoneum being tightly closed. However, the layers of the incision were only loosely closed, and dependent drainage down to the peritoneum or rectus muscle was instituted. There were 97 examples (12.92 per cent) of secondarily formed pelvic abscesses that required drainage. Seventy-three (9.72 per cent) of these pelvic abscesses occurred in persons whose operations were performed through either midright rectus incisions or midline incisions.

Twelve (1.60 per cent) of the 97 patients with pelvic abscesses died; 10 (1.33 per cent) of these deaths were attributable to the non-muscle-splitting incisions. These pelvic abscesses, after their localization, were

TABLE 11.—*Percentage of Mortality per Age Group Compared With Previously Reported Series*

Age Groups (in Years)	Percentage of Mortality per Age Group	
	Average of 21,289 Cases Reported by Various Authors* Between the Years of 1931-1936	Collins, 1938, 751 Cases
1-10	10.6	10.8
11-20	2.66	4.3
21-30	4.0	4.3
31-40	6.7	4.9
41-50	11.14	14.1
51-60	11.70	25.0
61-70	21.65	50.0
71-80	57.2	40.0
81-90	100.0	....

* Bower (in 1931); Wevill and Wallace; Pattison; Pool; Boyce and McFetridge, and Rhodes, Birnbaum and Brown.

TABLE 12.—*Relationship of Duration of Symptoms Before Operation With Percentage of Mortality in Acute Appendicitis; Comparison of This Series With Others*

Time in Hours	Author, Year and Number of Cases Reported					
	Bower, 1931, 5,121	Boland, 1932, 4,270	Maes and Others, 1935, 2,295	Pattison, 1936, 1,166	Boyce and Others, 1936, 2,715	Collins, 1938, 751
0- 6	2.55	0.00	2.0	0.00	1.6	1.7
7-12		1.00				0.0
13-24		2.32	2.9	0.48	2.8	3.5
25-48	6.31	6.45	4.6	5.60	4.8	5.9
49-72	8.59	6.50	7.5		6.8	7.3
Over 72	11.83	8.00	12.3	9.80	15.7	13.2

TABLE 13.—*Mortality for the Surgical Treatment of Acute Appendicitis*

Author and Year	Total Cases	Total Deaths	Average Mortality, Percentage
Various authors,* 1931 to 1937	60,130	3,046	5.06
Collins, 1938	751	58	7.714

* Bower; Boland; Finney; Keyes; Reid; Wevill and Wallace; Stanton; Harris; Cayford; Lied; Black; Merrell; Kline; Elken; Maes and McFetridge; Krasvoselskiy; Greiner; Schroder and Steinel; Lamon; Liveland; Hobler; Reid, Poer and Merrell; Nuzum; Pattison; Cook and Beardsley; Pool; Boyce and McFetridge; Rhodes, Birnbaum and Brown; Jensenius; Sperling and Myrick, and Holder and Wells.

drained, wherever possible, either through the anterior rectal wall in men or through Douglas' cul-de-sac in women. No permanent fistulas resulted from such methods of drainage, and none of the patients died.

Eleven instances (1.46 per cent) of subdiaphragmatic abscesses were encountered and 4 of the patients affected died. There were 8 patients

(1.07 per cent) with multiple abscesses of the liver, all of whom died. Of 69 persons (9.19 per cent) with postoperative pneumonia, 9 (1.20 per cent) succumbed. Twenty-seven (3.6 per cent) of this group had had definite preoperative infections of the upper part of the respiratory tract. Among the 113 instances (15.05 per cent) of generalized peritonitis, there were 25 deaths (3.33 per cent).

TABLE 14.—*Mortality in Relation to Types of Abdominal Incision*

Authors	Year	Number of Cases	Paramedian and Midright Rectus	Muscle-Splitting and McBurney's
Taylor	1935	110	40.00	10.00
Reid and others	1936	2,035	9.50	5.40
Pattison	1936	1,181	6.4	0.6*
Collins	1938	751	6.12	1.60

TABLE 15.—*Mortality in Various Types of Acute Appendicitis; Comparison With Other Series Reported in the Literature*

Author and Year	Total Cases	Average Incidence in Percentage		
		Acute Appendicitis with Nonperforated Appendix, Including Gangrenous Specimens	Acute Appendicitis, with Perforated Appendix, Abscess or Local Peritonitis	Acute Appendicitis, General Peritonitis
Various authors,* 1933 to 1937	18,899	1.45	6.24	28.57
Collins, 1938	751	3.09	6.41	22.12

* Finney; Keyes; Harris; Cayford; Haggard; Leid; Merrell; Eiken; Reid; Poer and Merrell; Lamon; Hobler; Pattison; Rhodes, Birnbaum and Brown, and Sperling and Myrick.

TABLE 16.—*Deaths Caused by Preoperative Catharsis; Comparison With Other Series*

Author and Year	Number of Cases	Average Percentage of Patients Having Had Laxatives	
		Among Total Postoperative Deaths	In Entire Series
Various authors,* 1931 to 1937	21,206	47.4	20.1
Collins, 1937	751	50.0 (29 cases)	18.2 (137 patients)

* Bower; Boland; Keyes; Merrell; Lamon; Reid, Poer and Merrell; Cook and Beardsley; Boyce and McFetridge, and Sperling and Myrick.

Thus, in the entire series of 751 cases, there were 58 deaths (7.72 per cent), as detailed in tables 9 to 13. However, only 12 (1.60 per cent) of the total of 58 deaths occurred in the cases in which muscle-splitting incisions were performed. (Consult table 15 for additional data.) Twenty-nine (3.86 per cent) of the 58 patients who died had taken one or more doses of laxative prior to entrance into the hospital. (See table 16 for further details.) This fact fully substantiates the

previous contentions of Bower as to the deadliness of preoperative catharsis in instances of acute appendicitis.

Some of the important features of the operative technic were briefly as follows: Less attention was paid to the day of the disease than to the condition of the patient. If the patient was growing steadily worse, exploration was usually believed to be indicated. If, on the other hand, the patient had a palpable mass in the lower part of the abdomen and was improving, surgical intervention was not employed at that time. Spinal anesthesia was used routinely unless some condition was present in the patient that served as a definite contraindication to its use. Every possible effort was made to drain extraperitoneally localized appendical abscesses, but such abscesses were never drained across the clean peritoneal cavity. Only localized appendical abscesses were drained by the use of simple Penrose drains constructed from rubber dam material.

If the appendix formed part of the wall of an abscess, if its removal would lead to the extensive breaking down of protective adhesions or if the patient was a poor risk, operative time was not wasted, nor was the life of the patient placed in additional jeopardy by foolish insistence on the completion of a difficult appendectomy. In such cases the patients were asked to return to the hospital for the performance of an interval appendectomy 10 weeks after their dismissal. All pelvic abscesses were drained, if possible, through either Douglas' cul-de-sac or the anterior rectal wall. The stump of the appendix was not inverted, and no reason has been encountered to regret the use of this simpler technic. I employ muscle-splitting incisions as a matter of routine whenever possible, because I believe that the use of such incisions insures a definite lowering of both the operative mortality and the postoperative morbidity. In no instance was a tight reapproximation of the edges of the skin permitted. Skin clips were used by preference. At the first sign of infection of the wound, adequate through and through drainage was instituted, both to hasten the final healing of the incision and to prevent the onset of extensive phlegmons of the anterior abdominal wall, which are often of anaerobic origin.

All patients were placed in Fowler's position postoperatively, and the principles of Ochsner were closely followed. Nothing was given by mouth until flatus was passed, abdominal distention had disappeared and the temperature had fallen toward normal. In the interim, fluids were usually administered by hypodermoclysis, or, if necessary, by the intravenous route, usually in daily amounts of 2,000 cc. No patient was allowed to be out of bed until the incision was healed and the temperature had been afebrile for 4 days. Before dismissal from the hospital, a rectal examination was mandatory to exclude the possibility of the

presence of a silent secondary pelvic abscess. All patients were closely followed for a month after dismissal from the hospital, and after that at less frequent intervals.

CONCLUSIONS

Seven hundred and fifty-one instances of acute retrocecal appendicitis have been reported. The mortality for this series was 7.72 per cent. Comparisons between acute retrocecal appendicitis and the ordinary acute types reported in other series lead one to the conclusion that this is the most dangerous form of appendicitis. Data from this material showed that muscle-splitting incisions are the safest and that they constitute a significant factor in the reduction of both the operative mortality and the postoperative morbidity.

Women appear to withstand the ravages of appendicitis better than men. The deadliness of preoperative self catharsis has been again substantiated. Appendixes assume a fixed retrocecal position from adhesions produced by previous attacks of acute appendicitis. Sixty-two per cent of the patients of this series had had previous similar attacks. The mortality appreciably increased in the first and after the fifth decade of life. Forty-three per cent of the total deaths were due to generalized peritonitis. Fifty per cent of those who died had had preoperative self catharsis. A plea is earnestly made for the recognition of this dangerous and atypical variety of appendicitis.

BIBLIOGRAPHY

Black, cited by Haggard.

Boland, Frank K.: Results in Treatment of Acute Appendicitis: Review of 4,270 Cases in Atlanta, J. A. M. A. **99**:443-448 (Aug. 6) 1932.

Bower, John O.: Acute Appendicitis: A Survey of Its Incidence and Care in Philadelphia, J. A. M. A. **96**:1461-1465 (May 2) 1931.

The Mortality of Acute Appendicitis: A Report of the Progress Made in a Campaign for Its Reduction in Philadelphia, ibid. **99**:1765-1768 (Nov. 19) 1932.

Boyce, Frederick F., and McFetridge, Elizabeth M.: The Essential Clinical Considerations of Acute Appendicitis, Internat. S. Digest. **22**:195-213 (Oct.) 1936.

Brunn, Harold: Acute Pelvic Appendicitis, Surg., Gynec. & Obst. **63**:583-592 (Nov.) 1936.

Cayford, E. Howard: Acute Appendicitis: A Review of Six Hundred and Fourteen Cases with Special Reference to Drainage, Canad. M. A. J. **32**:259-262 (March) 1935.

Chopra, B. L.: Three Cases of Adherent Retrocaecal Appendix, Indian M. Gaz. **71**:467-468 (Aug.) 1926.

Collins, Donald C.: The Length and Position of the Vermiform Appendix, Ann. Surg. **96**:1044-1048 (Dec.) 1932.

Mechanism and Significance of Obliteration of the Lumen of the Vermiform Appendix, ibid. **104**:199-211 (Aug.) 1936.

Cook, Charles O., and Beardsley, J. Murray: Appendicitis: Diagnosis, Treatment and End Results, Rhode Island M. J. **19**:69-82 (May) 1936.

Davis, C. R.: Critical Analysis of Thirty-Five Deaths Following Appendicitis, Am. J. Surg. **29**:369-372 (Sept.) 1935.

Eiken, T.: Mortality in Appendectomy, Ugesk. f. læger **97**:485-488 (April 25) 1935; abstracted, Brit. M. J. **2**:21 (Aug. 10) 1935.

Finney, J. M. T., Jr.: Appendicitis: Some Observations Based on a Review of 3,913 Operative Cases, Surg., Gynec. & Obst. **56**:360-365 (Feb. 15) 1933.

Greiner, W.; Schrader, R., and Steinel, C.: Acute Appendicitis and Its Complications in the Göttingen Clinic Between 1912 and 1934, Beitr. z. klin. Chir. **162**:426 (Oct. 23) 1935; abstracted, J. A. M. A. **106**:81-82 (Jan. 4) 1936.

Haggard, William D.: Appendicitis: An Analysis of 3,344 Cases with Remarks on the Delayed Operation in Delayed Appendicitis, Am. J. Surg. **28**:71-77 (April) 1935.

Harris, Charles H.: A Review of 1,007 Cases of Appendicitis with a Survey of Some of the Causes of Morbidity and Mortality, Texas State J. Med. **30**:579-583 (Jan.) 1935.

Hobler, Lawrence L.: Appendicitis: An Analysis of 4,833 Cases, Ann. Surg. **103**:86-96 (Jan.) 1936.

Holder, Hall G., and Wells, John Thorpe: Survey of Appendicitis in San Diego, Surg., Gynec. & Obst. **64**:239-244 (Feb. 1) 1937.

Jensenius, H.: Mortality from Appendicitis, Ugesk. f. læger **98**:1085-1089 (Nov. 5) 1936; abstracted, Brit. M. J. **1**:13-14 (Jan. 23) 1937.

Keyes, E. Lawrence: Death from Appendicitis: The Mortality from Appendicitis and the Causes of Death Following Appendicitis, Ann. Surg. **99**:47-68 (Jan.) 1934.

Kline, L. B.: Operative Mortality in Acute Appendicitis, Mil. Surgeon **76**:246-250 (May) 1935.

Krasnoselskiy, M. V.: Operative Treatment of Appendiceal Peritonitis Without Drainage, Vestnik khir. **40**:193, 1935; abstracted, J. A. M. A. **105**:2119 (Dec. 21) 1935.

Lamon, John Daniel: Acute Suppurative Appendicitis: Report of Two Hundred and Six Consecutive Cases, Southwestern Med. **20**:5-7 (Jan.) 1936.

Lascaux, Robert: L'appendicectomie dans les appendicites rétro-caecales, Presse méd. **42**:685-687 (April 28) 1934.

Lesser, Albert, and Goldberger, Harold A.: The Blood Sedimentation Test and Its Value in the Differential Diagnosis of Acute Appendicitis, Surg., Gynec. & Obst. **60**:157-166 (Feb. 1) 1935.

Lied, cited by Haggard.

Loveland, John E.: A Lowered Death Rate for Acute Appendicitis, Am. J. Surg. **31**:87-91 (Jan.) 1936.

Maes, Urban, and McFetridge, Elizabeth M.: Acute Appendicitis at the Extremes of Life, Based on an Analysis of Four Hundred and Twenty-Six Cases in Children Under Twelve and Two Hundred and Twenty-Four Cases in Adults Over Forty Years of Age, South. Surgeon **4**:422-437 (Dec.) 1935.

Meagher, J. L.: A Retrocaecal Appendix, M. J. Australia **1**:339 (March 16) 1935.

Merrell, Paul: A Statistical Study of 2,921 Cases of Appendicitis, J. Med. **16**:124-127 (May) 1935.

Meyer, Karl A., and Spivack, Julius L.: On the Relationship Between Retrocecal Appendix and Lane's Kink and Its Surgical Significance, Am. J. Surg. **25**: 12-13 (July) 1934.

Nuzum, T. W.: Appendicitis: A Study of 1,734 Cases, Wisconsin M. J. **35**:109-111 and 162 (Feb.) 1936.

Pattison, Arthur C.: Factors in the Mortality of Acute Appendicitis, Ann. Surg. **103**:362-274 (March) 1936.

Pool, R. M.: Acute Appendicitis: Report of Seven Hundred and Fifty-Seven Operated Cases, Am. J. Surg. **32**:469-473 (June) 1936.

Reducing the Mortality of Acute Appendicitis, editorial, J. A. M. A. **106**:2160-2161 (June 20) 1936.

Reid, Mont R.: Some Remarks on the Operative Procedures for Appendicitis, Surg., Gynec. & Obst. **59**:529-531 (Sept.) 1934.

——Poer, D. Henry, and Merrell, Paul: A Statistical Study of 2,921 Cases of Appendicitis, J. A. M. A. **106**:665-669 (Feb. 29) 1936.

Rhodes, George K.; Birnbaum, Walter, and Brown, Maurice J.: Acute Appendicitis: A Clinical Review of One Thousand Consecutive Cases, California & West. Med. **45**:458-463 (Dec.) 1936.

Sperling, L., and Myrick, J. C.: Acute Appendicitis: A Review of Five Hundred and Eighteen Cases in the University of Minnesota Hospitals from 1932-1935, Surgery **1**:255-264 (Feb.) 1937.

Stanton, E. M.: Acute Appendicitis: A Study of the Correlation Between the Time of Operation, the Pathology, and the Mortality, Surg., Gynec. & Obst. **59**:738-744 (Nov.) 1934.

Stein, Jacob L., and Wolfson, William Leon: Retrocecal Gangrenous Appendicitis, Am. J. Dis. Child. **48**:580-585 (Sept.) 1934.

Taylor, Hermon: Appendicitis in the Aged, Lancet **2**:937-938 (Oct. 26) 1935.

Wakeley, Cecil P. G.: The Position of the Vermiform Appendix as Ascertained by an Analysis of 10,000 Cases, J. Anat. **67**:277-283 (Jan.) 1933.

Wangensteen, O. H.: The Mechanism of the Vermiform Appendix—a Potential "Closed-Loop," editorial, Surg., Gynec. & Obst. **62**:1020-1022 (June) 1936.

Wevill, L. B., and Wallace, H. L.: Acute Appendicitis: A Statistical Survey of 8,000 Cases, Edinburgh M. J. **41**:557-582 (Oct.) 1934.

Wilkie, D. P. D.: Observations on Mortality in Acute Appendicular Disease, Brit. M. J. **1**:253-255 (Feb. 14) 1931.

Wolford, Thomas: Retrocecal Appendices, New Orleans M. & S. J. **87**:622-624 (March) 1935.

FRESH FRACTURE OF THE OS CALCIS

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A broken leg as a rule causes too great a disability to allow a mistaken diagnosis. Not so with many fresh fractures of the os calcis. If the damage is severe enough the physician's attention is focused and his diagnostic curiosity challenged to the point that early roentgenograms are generally taken. Thus, a "sprain" turns out to be a crushing fracture of the heel bone. Too often, even in the larger centers, the ancient empirical form of treatment, consisting of "rest in an encasement until consolidation has arrived," is applied. I wish to advocate, briefly, a manner of treating fresh fracture of the os calcis by closed reduction, modeling the fragments by force and encasing the restored foot until healing takes place.

HISTORICAL TREATMENT

In 1720 Garangeot, of France, first described accurately the "smash fracture." Petit and Desault, his countrymen, reported their cases in the same year and advised "rest until fragments had consolidated." Bilgner, of Germany, in 1720 added to the literature a good clinical description of the injury. For one hundred years no one propounded any change in the treatment, though a number of papers were published. The surgeon of that time appeared contented to describe in great detail every clinical variation of the condition and was not much interested in treatment. Cooper, in England, in 1835 recommended dressings of egg albumin and cotton lint unless the extremity appeared gangrenous; if it was gangrenous he amputated. Pott sawed off the ends of the bone in all cases of compound fracture to prevent tetanus and so, of course, sawed away the heel bone when it was badly damaged. Mutilation and frequent deaths followed, as may be imagined. In 1839 Norris, the first American to report his results, followed the same rugged form of treatment. The first classic description and discussion of the mechanics involved in the production of this type of fracture were written by Nadal and Malgaigne in 1843, in Paris. Bérard in 1845, Bauchet in 1853 and Demarquay in 1854 advised the use of lint and cottage cheese encasement.

Clark, an American, in 1855 applied traction to the posterior fragment by means of a pulley apparatus; the exact method is not clear from his writings. He then used gutta-percha splints. Every one feared the

open approach to reduction until Charles Bell, of Edinburgh, in 1882 made it an accomplished fact in the case of a compound fracture. After reduction a lint packing treated with phenol was instilled into the wound, with "rest until consolidation took place" completing the treatment. Paszkowski in 1880 used the first encasement of plaster of paris. Bailey, of Oregon, in one of the first American medical journals, recommended "elevation, lotions, saline cathartics and no splints." He added that the foot is always stiff as an end result. The laborer's compensation laws of Europe, developed at about this time, caused an increased interest in fractures, and fracture of the os calcis came in for its share. German surgeons provided most interesting contributions.

The greatest incentive for better methods of treatment came with the advent of the roentgen ray, in 1895. Desfosses reported the first roentgen study of fracture of the os calcis and described the tearing so often found at the attachment of the achilles tendon. Helbing, Neuschafer and Lemmen in 1900 manually manipulated the foot over a wooden wedge and then encased it in plaster. Lecocq, of Lille, used relaxing massage with the knee flexed. Gérard, of Paris, in 1902 reported the use of fixation by means of bands of plaster of lead oleate and silver wire. Most authorities advised early weight bearing, with daily massage and exercise.

All this work occurred "B.C.," before Cotton, who in 1908 first published his method, used more universally thereafter than any other. Cotton and Wilson described their method of breaking up impaction of fragments and reduction by means of molding manually, after traction of the posterior portion of the body had pulled down the rear fragment. Impaction was accomplished by means of a sharp blow with a mallet over a felt pad, directed against the point of the heel. Traction was accomplished by means of a steel sound passed behind the achilles tendon and over the calcaneum. Motion in the subastragalar point was made certain by manually moving the lower part of the foot laterally and then applying a plaster encasement. Years later Cotton modified the traction method by using ice tongs and weight traction, with the foot and leg in a Thomas splint when the fracture was stubborn. Pads were strategically placed beneath each malleolus for pressure, and the foot was turned up in plantar flexion with inversion. The encasement was removed in four weeks, and weight bearing was allowed, with a transverse arch pad in the shoe. Cotton has changed recently to more conservative ways, not allowing weight bearing before the eighth or tenth week.

Curiously, in 1933, in discussing fracture of the os calcis in one of the leading surgical systems, Cotton stated that nothing was done about treatment until 1907-1908. American surgeons regarded Cotton's method highly, and many reported their results with it. A number

modified the method by substituting tenotomy of the achilles tendon. Cotton in 1921, in another classic, using freehand drawings from his notebook, a form of illustration which always enlivens an otherwise staid manuscript, expressed the opinion that impaction is not altogether sound mechanically and directed attention toward the remodeling of fragments as perhaps the most important step. He said that in all cases arthrodesis is distinctly inadvisable and that he preferred to break up adhesions in the subastragalar joint and restore lateral mobility. He then permitted weight bearing after the eighth week.

The following outline, illustrated in figures 1 and 2, is a review of the manifold ways of treating fractures of the os calcis.

1905

Eisendrath: open reduction, fixation with kangaroo tendon, cast.

1908

Cotton and Wilson: closed reduction after breaking up of impaction with a mallet, traction on the achilles tendon, reimpaction, cast with felt pads beneath malleoli for compression and under longitudinal arch of foot.

1912

Van Stockum: open reduction with subastragalar arthrodesis, cast.

1913

Drewke: open reduction, cast.

Gelinsky: closed reduction, drill fixation from without, cast.

Leriche: open reduction, bone plates, steel screws for fixation, cast.

Soubeyran and Rives: primary astragalectomy.

1916

Lounsbury: Cotton's technic, occasionally open reduction, fixation with kangaroo tendon, tenotomy, cast.

Forrester: manual manipulation, tenotomy, cast (with the foot in plantar flexion over a roller bandage and the heel well down), fixation with kangaroo tendon if necessary.

1917

Bendixen: Cotton's technic, carpenter's clamp or fixation with kangaroo tendon, cast.

Cahill: Cotton's technic or fixation with nail, cast.

Magnuson: essentially Cotton's technic plus tenotomy, cast, then special shoe.

1918

Whiteside: open reduction, fixation with silver wire, tenotomy, cast.

1919

Davis: Cotton's technic.

1920

Foldes: Fischer's leaf spring apparatus for external traction for reduction and maintenance, pin through posterior fragment.

Mumford: essentially Cotton's technic, with reduction leverage over iron pipe and tenotomy.

Magnuson: former technic plus reduction leverage over wedge, cast, weight bearing in eight weeks.

1921

Beedle: gradual reduction by daily manipulations, followed by use of a cast.

Straus: Steiman pin through posterior fragment, traction and reduction on Hawley table, cast.

Moreau: subastragalar arthrodesis immediately, occasionally astragalectomy and once amputation of foot.

Magnuson: old fractures, new bed for peroneal tendons, wrenching of foot, cast, Thomas heel and felt arch support in shoe.

Cotton: former technic plus cast with pad beneath lateral malleolus.

1922

Kaess: two pin traction on posterior fragment in plaster boot, heel portion fenestrated.

1923

Becker: open reduction by forcible leverage, fixation by drill point, cast over all.

1924

Sneed: Cotton's technic plus tenotomy, occasionally removal of excess bone beneath lateral malleolus, with subastragalar arthrodesis (old fractures).

Elsner: closed reduction, fixation with nail, cast.

1925

Hall: essentially Cotton's technic plus tenotomy.

1926

Harding: cabinet maker's D clamp to reduce and mold fragments, traction by claw retractor buried above os calcis, all over wedge in sole of foot, cast, weight bearing after twelve weeks.

Brickner and Milch: essentially Cotton's technic plus walking iron in cast and early bivalving, with active motion and massage.

Conn: direct traction, tenotomy, cast, occasionally subastragalar arthrodesis.

Condit: Cotton's technic plus tenotomy.

Reich: triple arthrodesis (old fractures), cast for twelve weeks.

1927

Speed: open reduction, fixation with catgut or ivory peg, general preference for Cotton's technic for closed reductions.

Funsten: Cotton's technic plus tenotomy.

Wilson: open reduction plus subastragalar arthrodesis, cast.

1928

Masland: cast to ankle, cast over forepart of foot, then iron clamps to compress and reduce fractures, plus pads under joining of cast.

Glasson: Cotton's technic.

Bendixen: Cotton's technic plus tenotomy.

Lenormant, Wilmoth and Lecœur: open reduction, with elevation of astragalus, plus osteoperiosteal grafts from lateral malleolus to fill defect in os calcis, cast.

1929

Böhler: closed reduction, wooden wedge in sole of foot to break up impaction, heel forced down, iron clamp for compression, pin through tibia and pin through posterior fragment of os calcis, knee flexed 90 degrees in frame stirrup, counter-traction on tibia, traction on os calcis, first cast for from three to five weeks, second cast for from nine to fourteen weeks, with walking iron, arch support in shoe.

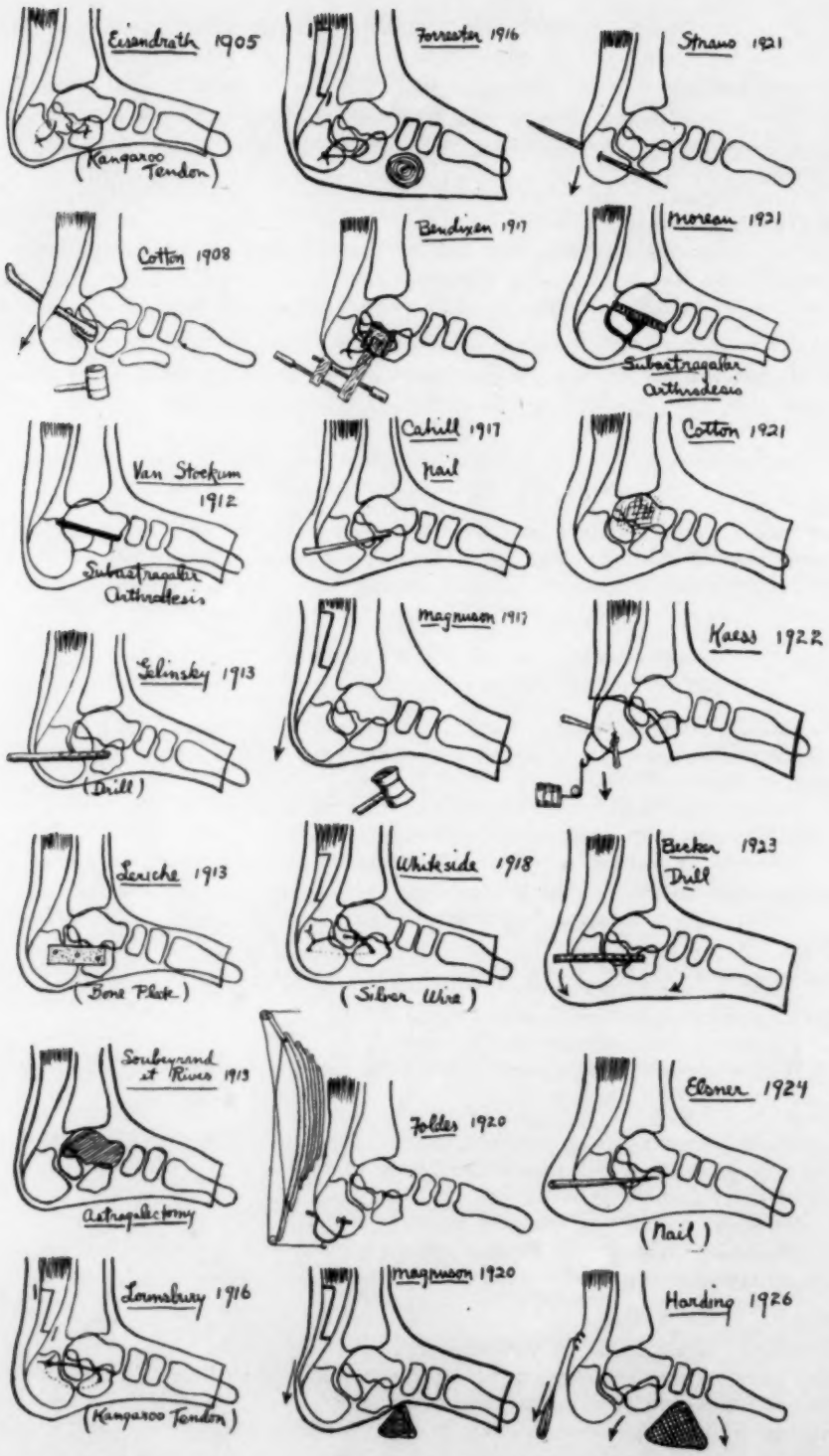

Fig. 1.—Pictorial review of the various methods of treating fracture of the os calcis.

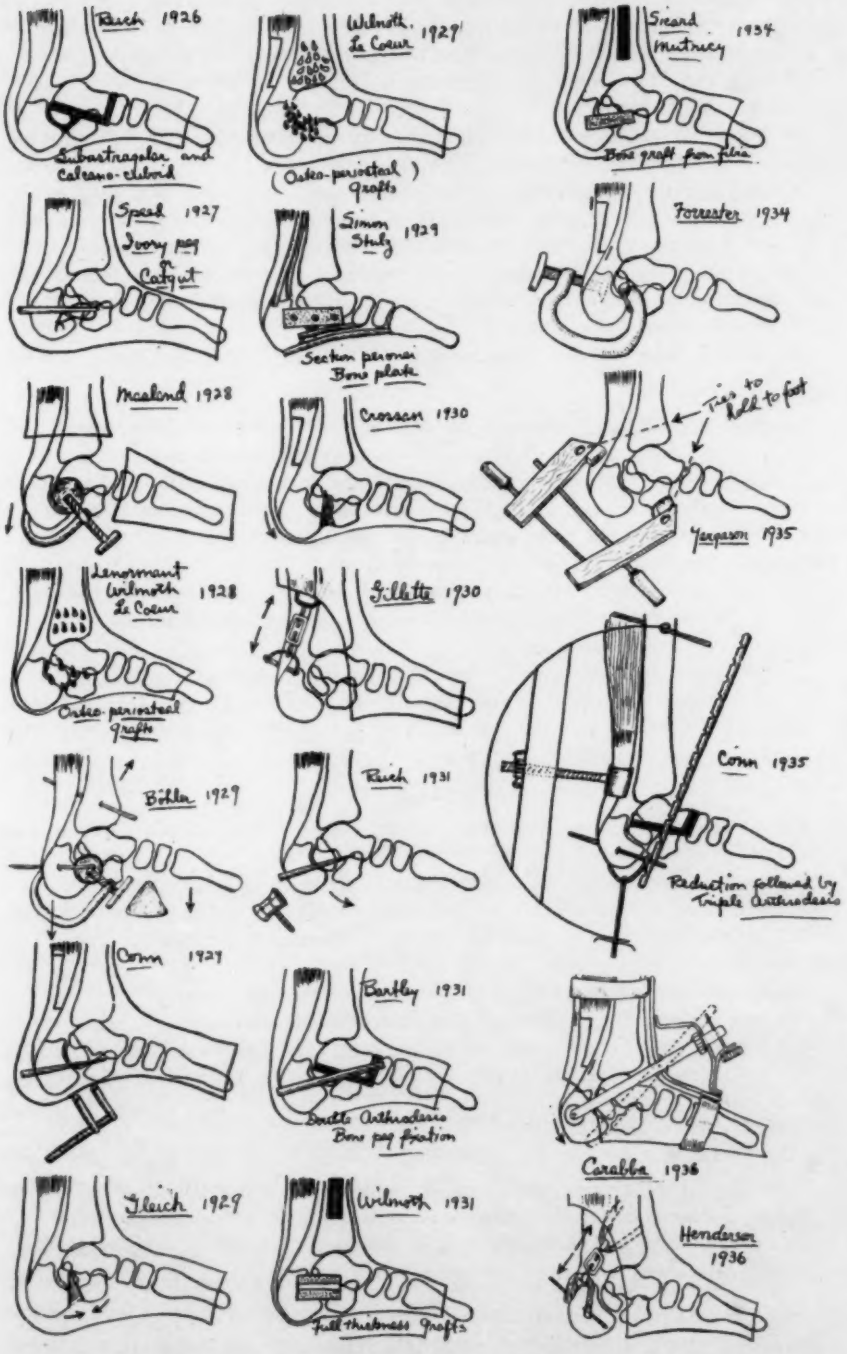

Fig. 2.—Pictorial review of the various methods of treating fracture of the os calcis, continued.

Conn: closed reduction, wrenching of foot, tenotomy, fixation with nail, cast, occasionally subastragalar arthrodesis.

Gleich: open reduction, wedge osteotomy, cast (fresh fractures).

Bérard and Mallet-Guy: open reduction, cast.

Schnek: Böhler's technic.

Wilmoth and Lecœur: open reduction, osteoperiosteal grafts, no cast for first week, then cast for twelve weeks.

Chatterton: Cotton's technic, with tenotomy.

Oudard and Le Bourgo: closed reduction by hand, cast, occasionally open reduction with fixative material and subastragalar arthrodesis.

Simon and Stulz: open reduction, sectioning of peroneal tendons, fixation with bone plate, suture tendons, cast; sequestration frequent result.

Schwartz: open reduction, cast.

Fontoynont and Estrade: cast alone.

1930

Bendixen: carpenter's clamp, with two blocks for compression, plus Cotton's technic, occasionally open reduction, fixation with kangaroo tendon, cast.

Crossan: triple arthrodesis, wedge osteotomy, tenotomy, cast (old fractures).

Simon and Stulz: open reduction, subastragalar arthrodesis, with packing in of bits of fragments cut from fracture site or medial malleolus (inner incisional approach), occasionally use of bone plates, screws.

Gillette: cast from toes to groin, fenestrated heel portion, turnbuckle component embedded in cast for push on pin above os calcis, behind achilles tendon, to accomplish reduction and fixation.

Pichon: open reduction, with osteoperiosteal grafts, cast.

Dieulafe: open reduction, fixation with silver wire, cast.

Hermann: Cotton's technic.

Dachtler: special fracture, cast alone.

1931

Reich: traction on heel plus impaction with mallet, occasionally fixation with nail, cast.

Bartley: open reduction, double arthrodesis, fixation with bone peg, cast.

White: rapid Böhler technic with patient on Hawley table, reduction accomplished immediately, cast wedged for further correction if necessary.

Mouchet and Allard: cast alone.

Didiée: fracture of apophysis, rare, manual replacement, cast.

Monod: cast for four weeks, then double arthrodesis.

Patel, open reduction, fixation with nail, cast, spontaneous arthrodesis desired.

Wilmoth: open reduction, grafts with full thickness from shaft of tibia for fixation, cast.

Böhler: former technic.

1932

Harding: former technic, occasionally fixation with screw or nail.

Reich: Cotton-Funsten combination, occasionally plus Böhler's technic.

Mouchet, Allard and Mégnin: special fracture of anterior portion of large apophysis, cast alone, with foot in marked dorsiflexion.

1933

Pierce: Cotton's technic, occasionally subastragalar arthrodesis.

Myers: impaction allowed to remain, cast for only three weeks, early weight bearing.

Funsten: compression bandage one week with elevation, Cotton's technic plus tenotomy, Thomas brace for early walking.

Moore: special fracture of tuber calcanei, rubber ball in cast under heel for pressure, cast for three weeks.

1934

Westhues: closed reduction, fixation with nail, cast for three weeks.

Sicard and Mutricy: open reduction, bone graft full thickness from tibia, tenotomy, cast after fifteenth postoperative day to permit swelling to subside.

Stewart: elevation, ice, closed reduction, cast, occasionally Böhler's technic, subastragalar arthrodesis if other methods failed.

Forrester: closed reduction, iron clamp for compression, tenotomy, walking iron incorporated in molded plaster splints.

Oserow: Wilmoth's technic.

Laffitte: open reduction, essentially the technic of Sicard and Mutricy.

1935

Yergason: closed reduction, molding of fragments with powerful clamp, special blocks on jaws, cast with walking irons.

Conn: modification of Böhler's technic, with extremity in a rocking apparatus, plus triple arthrodesis.

Mutricy: former technic preferred.

1936

Schofield: Böhler's technic.

Carabba: reduction device with pin through posterior portion; cast cut out around posterior portion, arm of device moves through arc and pushes down posterior fragment, cast then holds reduction by incorporating pin, pin unscrews in midportion for removal.

MacAusland: Böhler's technic.

Henderson: Modification of Gillette's technic, with cast; traction on posterior portion, with pin and turnbuckle incorporated in cast.

PROGNOSIS

Every one is agreed that fracture of the os calcis is the most disabling of all fractures involving the bones of the leg or the foot. Mercer said that it is the most disabling of all injuries. He stated that the injured person never completely recovers movements of inversion and eversion if the fracture involves the subtaloid joint. He gave as a prognosis from three to four years before complete recovery. Kessler suggested from three to four months of disability for minor fractures and from eight to twelve months for severe types. He did not hazard a statement of the usual percentage allowed for permanent partial disability in settled cases.

Harding gave five months for the average period of disability. Hermann reported 77 per cent of good results. Funsten allowed a period of disability of just under six months. Böhler did not come down to actual figures, but said that this fracture is a very disabling one. Forrester stated that the average period of disability for his patients was four and one-half months, with an average ultimate specific loss of 18

per cent. The general trend appears to be in the direction of less time lost from employment and much lower percentage allowances for permanent partial disability, in spite of a contrary opinion held by most insurance carriers.

TREATMENT

Eight patients with fresh fracture of the os calcis were treated by immediate reduction, with gas-oxygen anesthesia and a modified carpenter's clamp described by Yergason in 1935 being used. Such a clamp can be adapted for this use by fastening to the inner surface of each jaw, near the tips, a rounded wooden block about 4 cm. in diameter.

The blocks attached to the inner surface of each jaw are applied over thin felt to the lateral and the medial sides of the heel, and the clamp is screwed down by compression. One exerts tremendous force on the

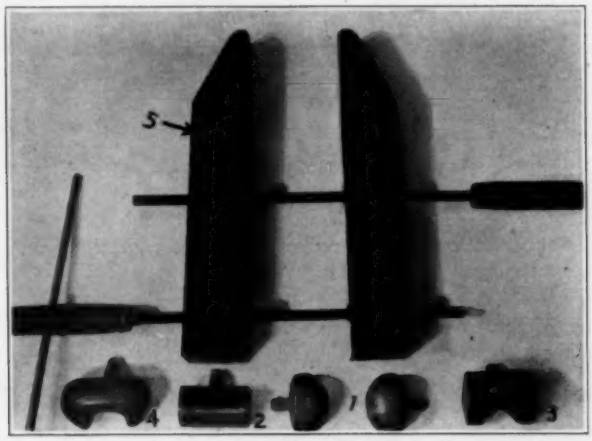

Fig. 3.—Clamp used in molding the fragments and reducing the fracture: *1*, hemispherical blocks for lateral modeling; *2*, oblong block used against plantar surface; *3*, grooved block to fit around the attachment of the achilles tendon over the tuberosity; *4*, semilunar block used beneath the lateral malleolus, and *5*, holes through the jaws to carry muslin ties to hold the clamp to the foot.

fragments in this manner, loosening the clamp and shifting its jaws over the surfaces to compress and mold the heel. A crescent-shaped block, easily made in one's workshop, is then substituted for one of the rounded blocks, and the extruded fragments are pressed into the body of the os calcis by applying the jaw bearing this crescent-shaped block beneath the lateral malleolus. In this manner all medial and lateral displacements can be corrected by pressure modeling with surprisingly little difficulty.

In cases of evulsion of the upper tuberosity, or fish mouth fracture, in which a fragment is pulled upward by the achilles tendon and there is plantar displacement of other fragments, the bones are molded forcibly

into position by applying the clamp at a right angle to the first position described. The crescent-shaped block on one jaw is placed over the attachment of the achilles tendon, and a square block, with edges smoothed, attached to the other jaw is applied on the plantar surface

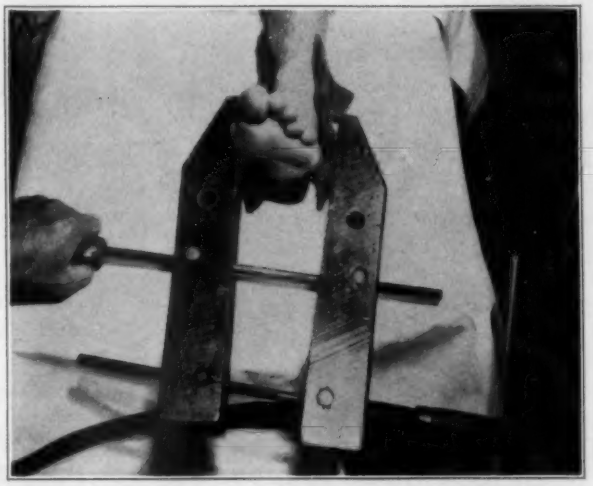

Fig. 4.—First position of application of force, with two hemispherical blocks, or jaws.

Fig. 5.—Second position, in which one hemispherical block and the semilunar block compress the fragment beneath the lateral malleolus that might extrude.

over the head of the os calcis. The clamp is then snugly adjusted while an assistant ties each jaw to the foot with a muslin loop passed around the foot, above and below the malleoli and through a hole in each jaw. These ties prevent the heel from sliding out of the clamp when the handles are screwed down and the jaws compressed. Thin felt must

always be used beneath the blocks and the muslin ties. The displaced fragments are then forced into position, and the posterior part of the os calcis is brought into normal relations with the head, so that the normal tuber joint angle is restored. This angle, described by Böhler, is made by the intersecting of a line drawn from the posterior-superior tuberosity of the os calcis to the superior-posterior portion of the astragalocalcaneal joint and a line drawn from the superior-posterior portion of the astragalocalcaneal joint and the anterior-superior projection of the head of the os calcis. This angle is normally between 25 and 35 degrees. Thus a tenotomy of the achilles tendon is unnecessary for reduction of the upper posterior fragment. This type of clamp is superior to Böhler's clamp because it permits application of much

Fig. 6.—Third position, in which blocks *2* and *3* are used to bring down the posterior tuberosity and raise the head of the os calcis. The clamp cannot get away from the foot because of the muslin ties.

greater force over all surfaces and allows the heel to be drawn down immediately, removing the necessity for continued traction.

Felt padding is then applied to the sole, extending around the heel and up to the knee. A snugly fitting plaster encasement is then adjusted from the toes to the upper part of the thigh; the knee is slightly flexed, with the foot in the position caused by slight talipes equinus, and a walking iron is incorporated. Elevation is insisted on for forty-eight hours, followed by walking on crutches, with weight bearing permitted after the tenth day. At the end of the second week the encasement is cut away above the knee. The plaster is removed at the end of the tenth week and physical therapy begun. Weight bearing is allowed at the end of the twelfth week, with a longitudinal arch support, made of firm felt or metal, in the shoe.

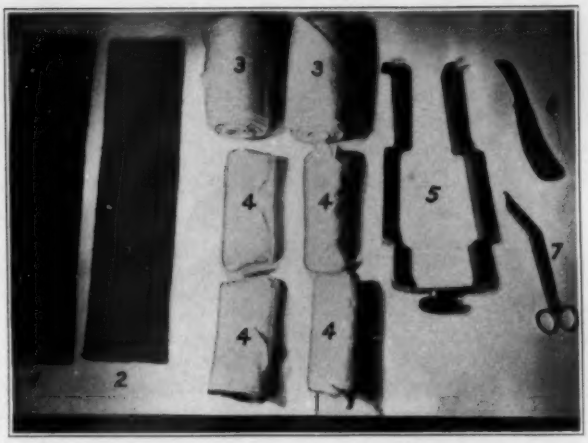

Fig. 7.—Material used in applying the walking cast: *1* and *2*, long strips of black felt; *3*, sheet wadding; *4*, plaster of paris; *5*, aluminum walking iron, and *6* and *7*, plaster knife and scissors.

Fig. 8.—Application of the walking cast; manner of padding beneath the walking iron.

REPORT OF CASES

CASE 1.—P. M., a man aged 52, fell 25 feet (7.6 meters), landing obliquely on one foot and crushing completely the right os calcis; the tuber joint angle was obliterated. Reduction was accomplished by molding the posterior portion of the foot with a special clamp, and immobilization was accomplished by applying a plaster of paris encasement, which was worn for ten weeks. Weight bearing was permitted after the fourteenth week, and the injured man returned to work after the twenty-fourth week.

CASE 2.—D. P., a man aged 42, jumped from a height of 16 feet (4.9 meters), landing on a hard concrete surface and suffering a compression fracture of the

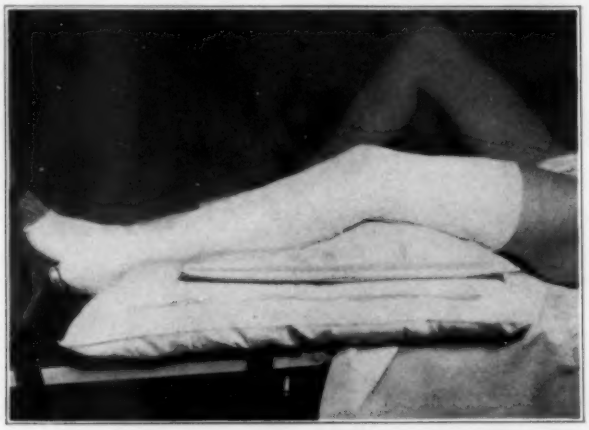

Fig. 9.—Walking cast completed and in position for drying.

Fig. 10.—Oxford worn, showing the type of leather and the longitudinal arch support of felt.

right os calcis. The tuber joint angle was obliterated, and the fragments were markedly displaced. Reduction of the fracture was accomplished by molding with special clamps while the patient was under general anesthesia. The tuber joint angle was restored, and a plaster of paris encasement incorporating a walking iron was applied. This was removed after the ninth week, and weight bearing was permitted after the eleventh week. The injured man returned to work after the thirteenth week. A spontaneous subastragalar arthrodesis materially aided his early recovery.

CASE 3.—H. B., a man aged 39, fell through the planking of a scaffold from a height of 12 feet (3.6 meters), fracturing his right os calcis. The fragments

were moderately displaced, so that a relatively small tuber joint angle resulted. Reduction was accomplished by molding with special clamps, and immobilization was accomplished by the application of a plaster of paris encasement. A walking block was incorporated in the plaster under the heel, a simple modification of a walking cast. This was removed after the eighth week, and weight bearing was permitted after the tenth week. The injured man returned to work after the seventeenth week, with all motion of the ankle and the foot completely restored.

CASE 4.—E. H., a man aged 25, fell through a scaffold onto a concrete surface 20 feet (6 meters) below. He suffered a comminuted fracture of the left os calcis, with moderate displacement, although the tuber joint angle remained practically normal. Reduction was easily accomplished by molding with special clamps, and a plaster encasement incorporating a walking iron was then applied. This encasement remained on for five weeks, and weight bearing was permitted after the sixth week. Pain was then confined about the subastragalar joint and proved intractable. A double arthrodesis was performed on the subastragalar joint, after which immobilization was maintained for eight more weeks. Two weeks after the removal of the plaster boot, weight bearing was permitted. The injured man returned to work after the twenty-fourth week.

CASE 5.—B. H., a man aged 42, fell from a height of 6 feet (1.8 meters) and landed on both heels on hard frozen ground. He suffered an oblique comminuted fracture of the right os calcis, with moderate displacement, although the tuber joint angle remained practically normal. Only lateral compression was necessary to reduce the fragment, and a plaster boot was applied and maintained for eight weeks. Weight bearing was permitted after the tenth week, and the injured man returned to work after the twelfth week.

CASE 6.—H. R., a man aged 40, fell from a girder 25 feet (7.6 meters) in the air, doing a complete somersault as he descended and landed on his heels and buttocks. He suffered a comminution of the right os calcis, with impaction in a distorted position, and a marked increase in the tuber joint angle. The impaction was broken up, and the fragments were molded into position by means of special clamps. A walking cast was maintained for eight weeks, and weight bearing was permitted after the twelfth week. The injured man returned to work after the twentieth week, with an excellent anatomic and functional result.

CASE 7.—J. W., a man aged 48, was accidentally pushed from the third floor of a building under construction and fell 60 feet (18 meters) onto a concrete base. He landed flat on his back, fracturing his skull, the spinous processes of the first and second lumbar vertebrae, the rami of the ischium and the pubis on the left side of his pelvis and crushing, or mushrooming, his left os calcis. When his condition would permit, his left os calcis was rebuilt, special clamps being used, so that the tuber joint angle was normal, and a plaster of paris encasement was applied. The encasement was maintained for six weeks, after which motion was begun. After the sixteenth week, when his general condition permitted, weight bearing was allowed and practically no pain occurred. He eventually made a complete recovery, but he was given a permanent disability rating of 50 per cent loss of the use of his left foot. Postreduction subastragalar arthritis seemed to be the cause of his pain. He refused further surgical intervention. This case was not used to compute the average length of disability, because of its complications.

CASE 8.—F. S., a man, aged 32, was precipitated down a brick shoot from 60 feet in the air, landing on a broken pile of bricks. He suffered a comminuted fracture of both his left and his right os calcis. The fractures were reduced by means of special clamps, and walking plasters were applied and maintained

for ten weeks. After the twelfth week weight bearing was permitted. After the sixteenth week the patient disappeared from the city, and he was not heard of for two years. When he returned he showed complete recovery of his left foot, but he had moderate postreduction traumatic arthritis of his right subastragalar joint. A double arthrodesis was done, and weight bearing was permitted after the sixteenth week. This patient is still under treatment, but a satisfactory result seems apparent.

In these cases the average age was 39 and the average fall 28 feet (8.5 meters). The patients were all men. Six of the fractures were on the right side and three on the left. The average period of disability was eighteen weeks. In five cases no percentage disability was allowed.

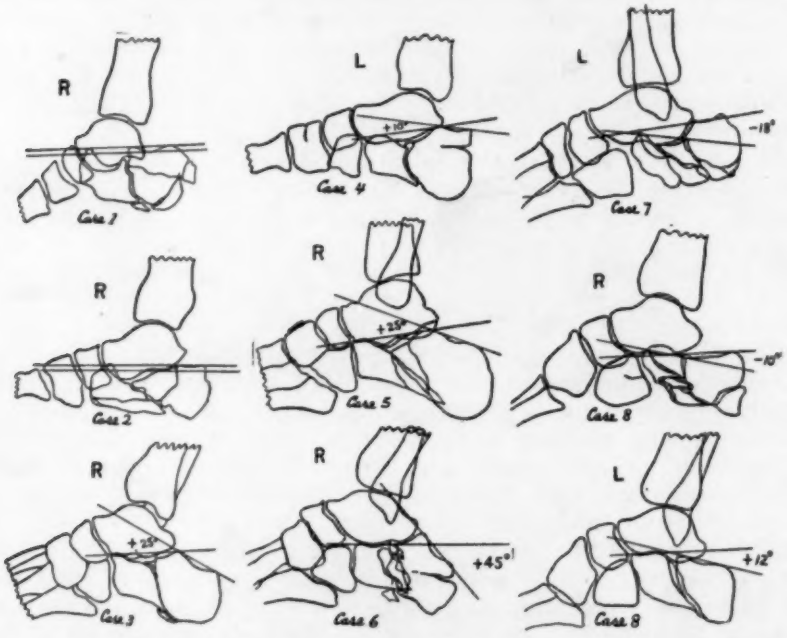

Fig. 11.—Tracings made from roentgenograms to illustrate the eight cases of fracture reported, with lines drawn to show the variety of tuber joint angles encountered.

In one case there was 10 per cent allowance and in another case 50 per cent. The treatment in one case is not completed.

RESULTS OF TREATMENT

Six of the 8 patients made a comparatively rapid recovery. One had post-traumatic arthritis in the subtaloid joint, refused arthrodesis and was given a 50 per cent disability allowance. The last patient disappeared for two years before treatment had been completed. When he returned his complaints were centered about the plantar surface of the

foot, and there was probably traumatic arthritis of the calcaneo-cuboid joint. The general architecture of the foot was excellent. This patient is still under treatment and was not considered when I determined the average length of disability.

While 8 cases are not many to report, there seems to be sufficient reason to offer this form of management of fracture for consideration. A crushed bone which is so accessible and which normally is so regular in shape should lend itself readily to molding if sufficient force can be applied without harm to the soft parts. The method is simple, effective, comfortable for the patient and physiologically reasonable and removes the necessity for tenotomy. Skeletal traction requires constant, skilled observation. The apparatus devised by Böhler requires rest in bed, is stationary and interferes with nursing care. This type of traction splint will some day be abandoned.

CONCLUSIONS

Fracture of the os calcis was formerly considered rare because it was not recognized. In 1835 Cooper detailed the best description and advocated "rest, elevation, until fragments consolidated." The use of this treatment persisted until the discovery of the roentgen ray, in 1895, after which manipulation and reduction were developed. The English propounded closed reduction, while the French turned to open operation with fixation and bone grafting. German and Austrian surgeons developed mechanics of the fracture and used skeletal traction for reduction, plus fixation. It was left for American surgeons to combine the many ways of approach and to work out the most generally acceptable methods. Cotton propounded a technic which holds today as the most universally used method.

Each surgeon has developed some slightly different attack, but all are together in their recognition of the difficulties in obtaining satisfactory results. Individual procedures are applicable to definite situations. The skill and judgment necessary for even passable results cannot be lightly considered. The more severe the fracture the more likely is a spontaneous arthrodesis to occur and the more excellent the result if the reduction is complete. Old, painful fractures call for a double or a triple arthrodesis. An occasional tenotomy may be necessary. Foreign body fixatives are used by some surgeons if comminution is not too great. Roentgen study must include at least four planes of visualization. An astragalectomy is never indicated. Skeletal traction in specialized apparatus gives good results if constant observation is possible. A restoration of the tuber joint angle, as described by Böhler, is essential. Closed reduction of a fresh fracture by forceful molding is recommended.

SUMMARY

The methods reported in the literature are discussed.

A pictorial method, affording a rapid review of technic, is utilized.

According to the literature, there appears to be a tendency to discontinue the use of internal fixatives and to depend on closed reduction, forcible molding of fragments and skeletal traction. An old, painful fracture is best treated by double or triple arthrodesis.

More recent writers report shorter periods of disability.

Eight cases are reported in which closed forceful molding by means of a clamp and blocks was used.

BIBLIOGRAPHY

Bilgner, 1720, cited by Nadal.

Desault, 1720, cited by Nadal.

Garangeot, 1720, cited by Nadal.

Petit, 1720, cited by Nadal.

Cooper, Samuel: Surgery, Philadelphia, Griggs & Elliot, 1835.

Bérenguier, A.: Fracture du calcanéum après une chute sur la plante des pieds, J. de chir. **1**:376, 1843.

Deville: Fracture par écrasement du calcanéum, Bull. Soc. anat. de Paris **18**:13, 1843.

Malgaigne, J. F.: Mémoir sur la fracture par écrasement du calcanéum, J. de chir. **1**:2, 1843.

Nadal, J.: Du mecanisme de la fracture du calcanéum, Thesis, Paris, no. 64, 1843.

Voillemier: Fracture par écrasement de deux calcanéums, resultat constaté 13 ans après l'accident, J. de chir. **1**:63, 1843.

Bérard, M.: Fracture du calcanéum par écrasement, Gaz. d. hôp. **7**:462, 1845.

More, quoted by Malgaigne: J. de chir. **3**:154, 1845.

Malgaigne, J. B.: Traité des fractures et des luxations, Paris, J. B. Baillière, 1847, vol. 1, p. 827.

Cooper, B.: Lectures on the Principles and Practice of Surgery, Philadelphia, Blanchard & Lea, 1852, p. 248.

Bauchet: Fracture par écrasement du calcanéum, Bull. Soc. anat. de Paris **28**: 215, 1853.

Demarquay: Fracture du calcanéum avec pénétration, Bull. Soc. de chir. de Paris **4**:483, 1854.

Clark, Le G.: Fracture of the Os Calcis, Lancet **1**:403, 1855.

Foucher: Fracture du calcanéum par écrasement, Bull. Soc. anat. de Paris **31**: 19, 1856.

Rémond, H. F.: Considérations sur la fracture par écrasement du calcanéum, Thesis, Paris, no. 236, 1857.

Cavasse: Fracture de deux calcanéums, Bull. Soc. anat. de Paris **33**:12, 1858.

Siredey, cited by Cavasse: Fracture du calcanéum par écrasement, Bull. Soc. anat. de Paris **33**:12, 1858.

Legouest, L.: Des fractures du calcanéum, Arch. gén. de méd. **2**:148, 1860.

Sonrier: Fracture du calcanéum par écrasement, Rec. de mém. de méd. mil. **20**: 466, 1868.

Polaillon: Calcanéum, Dict. encycl. d. sc. méd. **11**:606, 1870.

Pradel, L.: Considérations sur les fractures par écrasement du calcanéum, Thesis, Paris, no. 309, 1875.
Krabbel: Complizierte Fraktur des Calcaneus, Arch. f. klin. Chir. **23**:816, 1879.
Pinner, O.: Zur Casuistik der Calcaneusfrakturen, Deutsche Ztschr. f. Chir. **12**: 437, 1879.
Abraham, P. S.: Fracture of the Os Calcis, Dublin J. M. Sc. **70**:527, 1880.
Bailey, F. A.: Fracture of Os Calcis, Proc. Oregon M. Soc. **7**:68, 1880.
Bennett, E. H.: Fracture of the Os Calcis, Dublin J. M. Sc. **70**:526, 1880.
Paszkowski, V.: De la fracture du calcanéum par écrasement et des déformations consécutives, Thesis, Paris, no. 249, 1880, p. 64.
Bell, C.: Compound Fracture of the Os Calcis, Edinburgh M. J. **27**:1100, 1881-1882.
Fifield, W. C. B.: Fracture of the Os Calcis of Each Foot: Death from Embolism on the Twelfth Day; Autopsy, M. News, Philadelphia **42**:126, 1883.
Saussol, A.: Un cas de fracture du calcanéum par arrachement, Gaz. hebd. d. sc. méd. de Montpellier **5**:157, 1883.
Webb: Fracture of the Os Calcis, Australian M. J. **6**:362, 1884.
Desfosses, 1898, cited by Lecocq.
Tuffier, 1898, cited by Lecocq.
Helbing, 1900, cited by Gérard.
Lecocq, S. T.: Une méthode de traitement des fractures du calcanéum, Thesis, Lille, no. 470, 1900, p. 84.
Lemmen, 1900, cited by Gérard.
Neuschafer, 1900, cited by Gérard.
Gérard, L.: Fractures du calcanéum, Thesis, Paris, no. 342, 1902, p. 41.
Eisendrath, D. N.: Fracture of the Os Calcis, Ann. Surg. **41**:363, 1905.
Cabot, H., and Binney, H.: Fractures of Os Calcis and Astragalus, Ann. Surg. **45**:51, 1907.
Cotton, F. J., and Wilson, L. T.: Fractures of Os Calcis, Boston M. & S. J. **159**: 559, 1908.
Cotton, F. J.: Dislocations and Joint Fractures, Philadelphia, W. B. Saunders Company, 1910, pp. 589-621.
Van Stockum, A.: Operative Behandlung der Calcaneus- und Talusfractur, Zentralbl. f. Chir. **30**:1438-1439, 1912.
Drewke, W.: Frakturen des Calcaneus und ihre Behandlung, Inaug. Dissert., Berlin, 1913.
Gelinsky: Die Extensionbehandlung der Calcaneumfraktur und den Verletzungen der Mittelfussknoche, Zentralbl. f. Chir. **11**:809, 1913.
Leriche: Fracture compliquée du calcanéum; résection immédiate; guérison avec excellent résultat fonctionnel, Lyon méd. **120**:1185, 1913.
Soubeyran, P., and Rives, A.: Fractures du calcanéum, Rev. de chir., Paris **47**: 429, 1913.
Cotton, F. J.: Os Calcis Fractures, Ann. Surg. **64**:480, 1916.
——and Henderson, F. F.: Results of Fractures of the Os Calcis, Am. J. Orthop. Surg. **14**:290, 1916.
Forrester, C. R. G.: Prevention of Disability Following Fracture of Os Calcis, Illinois M. J. **30**:386, 1916.
Lounsbury, B. F.: Fracture of the Os Calcis, Surg., Gynec. & Obst. **22**:638, 1916.
Bendixen, P. A.: Fractures of the Os Calcis, J. Iowa M. Soc. **7**:140, 1917.
Cahill, G. F.: Fracture of the Os Calcis, Ann. Surg. **66**:711, 1917.
Koch, J. C.: Laws of Bone Architecture, Am. J. Anat. **21**:211, 1917.
Magnuson, P. B.: Fractures of the Os Calcis, J. A. M. A. **68**:530 (Feb. 17) 1917.

Whiteside, G. S.: A Case of Fracture of the Os Calcis, U. S. Nav. M. Bull. **12**: 267, 1918.

Davis, B. F.: Fracture of the Os Calcis, S. Clin., Chicago **3**:1307, 1919.

Glasson, C. J.: Case of Unusual Fracture of the Os Calcis, Arch. Radiol. & Electroth. **24**:228, 1919.

Foldes, D.: Fractures of the Patella, Os Calcis and Olecranon, Treated by Fischer's Apparatus, Surg., Gynec. & Obst. **30**:510, 1920.

Magnuson P. B., and Coulter, J. S.: Fracture of Both Os Calci, Internat. Clin. **2**: 224, 1920.

Mumford, E. B.: Fractures of Calcaneum, J. Indiana M. A. **13**:200, 1920.

Steindler, A.: Stripping of Os Calcis, J. Bone & Joint Surg. **2**:8, 1920.

Zur Verth, M.: Mechanik und System des Fersenbeinbrüches, München. med. Wchnschr. **39**:1115, 1920.

Ueber den Bruch der Fersenhöckers, Deutsche Ztschr. f. Chir. **153**:414, 1920.

Beedle, G. A.: Fractures of Os Calcis with Dislocation of the Astragalus, J. Missouri M. A. **18**:54, 1921.

Cotton, F. J.: Os Calcis Fractures, S. Clin. North America **1**:917, 1921.

Old Os Calcis Fractures, Ann. Surg. **74**:294, 1921.

Moreau, L.: Fractures directes du calcanéum, Paris méd. **11**:305, 1921.

Straus, D. C.: A New Method of Treating Recent Fractures of the Os Calcis, J. A. M. A. **77**:176 (July 16) 1921.

Kaess, W.: Die Behandlung der Fraktur des Calcaneuskörpers mittels Nagelextension, Zentralbl. f. Chir. **49**:1412, 1922.

Vulliet, H.: Une fracture inédite du calcanéum, Rev. méd. de la Suisse Rom. **42**: 815, 1922.

Becker, E.: Zur blutigen Behandlung der Fersenbeinbrüche, Zentralbl. f. Chir. **50**:262, 1923.

Outline of Treatment of Fractures, Arch. Surg. **6**:172 (Jan.) 1923.

Magnuson, P. B.: Operation for Relief of Disability in Old Fractures of the Os Calcis, J. A. M. A. **80**:1511 (May 26) 1923.

Elsner, J.: Die Osteotomie und zietweilige Nagelung des Calcaneus, bei blutigen Klumpfussoperationen, Zentralbl. f. Chir. **51**:429, 1924.

Morton, D. J.: Evolution of the Longitudinal Arch of the Human Foot, J. Bone & Joint Surg. **6**:56, 1924.

Mechanism of the Normal Foot and of Flat Foot, ibid. **6**:368, 1924.

Sneed, W. L.: A Report of Eight Cases of Severe Crushing Fractures of the Os Calcis, J. Bone & Joint Surg. **6**:157, 1924.

Hall, A. C.: Fractures of the Os Calcis, Am. J. Roentgenol. **14**:27, 1925.

Brickner, W. M., and Milch, H.: Fractures of the Os Calcis, Internat. Clin. **11**:253, 1926.

Condit, L. I.: Fractures of the Os Calcis, Surg., Gynec. & Obst. **42**:133, 1926.

Conn, H. R.: Fractures of the Os Calcis: Diagnosis and Treatment, Radiology **6**:228, 1926.

Harding, M. C.: Os Calcis Fractures: A New Method of Reduction, J. Bone & Joint Surg. **8**:720, 1926.

Reich, R. S.: Subastragaloid Arthrodesis in the Treatment of Old Fractures of the Calcaneus, Surg., Gynec. & Obst. **42**:420, 1926.

Funsten, R. V.: Fractures of the Os Calcis, J. Michigan M. Soc. **26**:102, 1927.

Malpas, P.: Prognosis of Fracture of the Os Calcis, Brit. M. J. **1**:510, 1927.

Speed, K.: New Type of Fracture of Calcaneus with Discussion of Common Injuries, S. Clin. North America **7**:583, 1927.

Wilson, P. D.: Treatment of Fractures of the Os Calcis by Arthrodesis of the Subastragalar Joint, J. A. M. A. **89**:1676 (Nov. 12) 1927.

Bendixen, P. A.: Fractures of Os Calcis, Surg. J. **34**:115, 1928.

Lenormant, C.; Wilmoth, P., and Lecœur, P.: À propos du traitement sanglant des fractures du calcanéum, Bull. et mém. Soc. nat. de chir. **54**:1353, 1928.

Masland, H. C.: A Reducing and Fixation Splint for Fractures of the Os Calcis, Internat. J. Med. & Surg. **41**:191, 1928.

Robinson, L. F.: A Review of Fractures of the Os Calcis: Report of Case, South. M. J. **21**:583, 1928.

Scudder, C. L.: Treatment of Fractures, ed. 10, Philadelphia, W. B. Saunders Company, 1928, pp. 715-724.

Steidler, A.: Stripping of Os Calcis, Surg., Gynec. & Obst. **47**:523, 1928.

Bérard, L., and Mallet-Guy, P.: À propos du traitement sanglant des fractures récentes du calcanéum, Lyon chir. **26**:453, 1929.

Böhler, L.: Behandlung der Fersenbeinbrüche, Chirurg **1**:733, 1929.
Behandlung der Fersenbeinbrüche, Arch. f. klin. Chir. **157**:723, 1929.

Chatterton, C. C.: Fracture of Os Calcis, Minnesota Med. **12**:521, 1929.

Conn, H. R.: Fractures of the Os Calcis, Ohio State M. J. **25**:629, 1929.

Fontoynont and Estrade: Un cas de fracture du calcanéum, Bull. Soc. path. exot. **22**:388, 1929.

Oudard and Le Bourgo: Les fractures du calcanéum méconnues, Arch. de méd. nav. **119**:95, 1929.

Schnek, F.: Zur Behandlung der Fersenbeinbrüche, Chirurg **1**:1123, 1929.

Schwartz, A.: À propos des fractures du calcanéum, Bull. et mém. Soc. nat. de chir. **55**:148, 1929.

Simon, R., and Stulz, E.: Le traitement sanglant des fractures par écrasement du calcanéum, Rev. de chir., Paris **67**:354, 1929.

Werner, A.: Ueber Fersenbeinbrüche durch Abscherung, Deutsche Ztschr. f. Chir. **221**:405, 1929.

Wilmoth, P., and Lecœur, P.: Le traitement opératoire des fractures sous-thalamiques du calcanéum, J. de chir. **33**:781, 1929.

Bendixen, P. A.: Fractures of the Os Calcis, Internat. J. Med. & Surg. **43**:258, 1930.

Crossan, E. T.: Fracture of the Tarsal Scaphoid and of the Os Calcis, S. Clin. North America **10**:1477, 1930.

Dieulafe, L., and Dieulafe, R.: Fracture de l'angle postéro-supérieur du calcanéum, Arch. franco-belges de chir. **32**:448, 1930.

Gillette, E. P.: An Apparatus for Treatment of Fractures of the Os Calcis, J. Bone & Joint Surg. **12**:670, 1930.

Hermann, O. J.: Fractures of the Os Calcis, New England J. Med. **202**:705 (April 10) 1930.

Pichon, R.: Fractures du calcanéum, Gaz. d. hôp. **103**:277, 1930.

Simon, R., and Stulz, E.: Operative Treatment of Compression Fractures of the Calcaneum, Ann. Surg. **91**:731, 1930.

Bartley, S. P.: Treatment of Fractures of the Os Calcis, S. Clin. North America **11**:637, 1931.

Böhler, L.: Diagnosis, Pathology and Treatment of Fractures of Os Calcis, J. Bone & Joint Surg. **13**:75, 1931.

Dachtler, H. W.: Fractures of the Anterior-Superior Portion of the Os Calcis Due to Violence, Am. J. Roentgenol. **25**:629, 1931.

Didiée, J.: Fracture parcellaire isolée du calcanéum, Rev. d'orthop. **18**:250, 1931.

Monod, R. C.: Traitement des fractures du calcanéum, Bull. méd., Paris **45**: 756, 1931.

Mouchet, A., and Allard: Fracture incomplète du bec de la grande apophyse du calcanéum, Ann. d'anat. path. **8**:1268, 1931.

Patel, M.: Fracture du calcanéum par écrasement; arthrodèse sous-astragalienne, Lyon chir. **28**:113, 1931.

White, J. W.: Fractures of the Os Calcis, South. Med. & Surg. **93**:184, 1931.

Wilmoth, P.: Traitement de fracture du calcanéum, J. de méd. et chir. prat. **102**: 328, 1931.

Davenport, C. B.: Growth of the Human Foot, Am. J. Phys. Anthropol. **17**: 167, 1932.

Harding, M. C.: Fractures of the Os Calcis, California & West. Med. **36**: 319, 1932.

Kessler, H. H.: Accidental Injuries, Philadelphia, Lea & Febiger, 1932.

Mercer, W.: Orthopedic Surgery, London, Edward Arnold & Co., 1932.

Mouchet, A.; Allard, and Mégnin, J.: Une fracture rare du calcanéum. (La fracture du bec de la grande apophyse), Rev. d'orthop. **19**:633, 1932.

Reich, R. S.: End Results in Fractures of the Calcaneus, J. A. M. A. **99**:1909 (Dec. 3) 1932.

Schwan: Einiges ueber Calcaneusbrüche, Med. Welt **6**:562, 1932.

Cotton, F. J.: Fractures, in Lewis D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1933, vol. 2, chap. 4, pp. 175-180.

Funsten, R. V.: A New Treatment for Fractures of the Os Calcis, Virginia M. Monthly **60**:265, 1933.

Keith, A.: Human Embryology and Morphology, ed. 5, Baltimore, William Wood & Company, 1933.

Moore, G. E.: Fractures of the Tuber Calcanei Involving the Medial and Lateral Processes, Surg., Gynec. & Obst. **57**:400, 1933.

Myers, G. P.: Fractures of the Os Calcis, Internat. J. Med. & Surg. **46**:69, 1933.

Pierce, F. E.: Disabilities Resulting from Fractures of the Os Calcis and Remedial Measures for Their Prevention, Internat. J. Med. & Surg. **46**: 522, 1933.

Clarkson, W., and Barker, A.: Fractures of the Os Calcis and Astragalus, South. M. J. **27**:397, 1934.

Forrester, C. R. G.: Acute Fractures of the Os Calcis, Am. J. Surg. **25**:404, 1934.

Küntscher, G.: Die Darstellung des Kraftfusses im Knochen, Zentralbl. f. Chir. **61**:2130, 1934.

Laffitte, H.: Résultats de trois fractures du calcanéum traitées par la greffe osseuse, Bull. et mém. Soc. nat. de chir. **60**:1348, 1934.

Oserow, A. D., and Ssolondsj, A. N.: Operative Treatment of Impacted Fractures of the Os Calcis, Sovet. khir. **6**:284, 1934.

Schindler, E.: Fractures of the Calcaneus in the Leipzig University Surgical Clinic (1926-1932), Dissert., Leipzig, 1934.

Sicard, A., and Mutricy, H.: À propos du traitement chirurgical des fractures sous-thalamiques du calcanéum, J. de chir. **43**:374, 1934.

Stewart, J. E.: Fractures of the Os Calcis, South. M. J. **27**:711, 1934.

Westhues: Zur Behandlung der Calcaneusfraktur, Zentralbl. f. Chir. **61**:2231, 1934.

Elftman, H., and Manter, J.: Chimpanzee and Human Feet in Bipedal Walking, Am. J. Phys. Anthropol. **20**:69, 1935.

Groves, E. W. H.: Organization of Treatment of Fractures, Brit. M. J. **1**: 813, 1935.

Mutricy, H.: Fractures du calcanéum; le probleme therapeutique qu'elles posent, Paris, Louis Arnette, 1935.

Willis, T. A.: Function of the Long Plantar Muscles, Surg., Gynec. & Obst. **60**:150, 1935.

Yergason, R. M.: A Simple Clamp for Reducing Fractures of the Calcaneum and Tibial Tuberosities, J. Bone & Joint Surg. **17**:225, 1935.

Carabba, V.: Apparatus for Treatment of Fractured Os Calcis, Am. J. Surg. **33**:53, 1936.

Henderson, M. S.: Fractures of the Os Calcis, Surg., Gynec. & Obst. **63**:782, 1936.

MacAusland, W. R.: Treatment of Fractures of the Os Calcis by Pin Traction, Surg., Gynec. & Obst. **63**:506, 1936.

Schofield, R. O.: Fractures of the Os Calcis, J. Bone & Joint Surg. **18**:566, 1936.

COLLECTION OF AIR IN THE RIGHT SUBDIAPHRAGMATIC SPACE

WITH SPECIAL REFERENCE TO HEPATODIAPHRAGMATIC INTERPOSITION OF THE COLON

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In the past decade, surgical literature has been replete with reports on eventration and on hernia of the diaphragm as well as on the role of the diaphragm in the surgical treatment of pulmonary tuberculosis, especially phrenicectomy and artificial pneumothorax. Articles in which the roentgenologic diagnosis of subphrenic abscess has been discussed are also available. There is, however, a dearth of material bearing on the significance of collections of air or gas under the diaphragm.

The presence of air in the subdiaphragmatic space is particularly pertinent when it is demonstrated roentgenologically to be on the right side, that is, between the diaphragm and the liver. In the left subphrenic area gas may be found in the cardiac end of the stomach (*Magenblase*) and in the splenic flexure of the colon, both of which lie directly beneath the leaf of the diaphragm. This is usually of no clinical importance. On the other hand, when gas or air occupies the space between the diaphragm and the liver, the finding signifies the presence of a pathologic condition or of some abnormality.

It is generally known that the right cusp of the diaphragm lies in direct contact with the superior surface, or dome, of the right lobe of the liver. During respiration in the normal state, the diaphragm and the liver move in unison. In a roentgenogram, their shadows merge and have the same density, so that one cannot be distinguished from the other. The histologic elements, such as the areolar tissue, the few blood vessels and the ligamentous attachment of the liver, that occupy the subphrenic space cast no differentiating shadows on the film. However, when this region is involved in a pathologic process, such as the presence of gas, fluid or tumefaction, which may cast a shadow of greater or less density than those of the liver and the diaphragm, or which by its presence may displace either one or both of these structures, a subdiaphragmatic lesion becomes evident in the roentgenogram. With the accumulation of air or gas in the subphrenic area, the potential space between the liver and the diaphragm becomes real. Gas is an excellent contrast medium, and by its presence the dia-

phragmatic dome and the superior surface of the liver are caused to stand out in silhouette.

CAUSES FOR THE PRESENCE OF SUBDIAPHRAGMATIC COLLECTIONS OF AIR

Air may be introduced into the subphrenic space either accidentally or for diagnostic purposes. In collapse therapy for tuberculosis or in the performance of an induced pneumothorax for diagnostic purposes, the needle may occasionally be introduced too low in the chest, so that the diaphragm is perforated and air is accidentally injected subdiaphragmatically before the operator is aware of the position of the needle (fig. 1). At times air is introduced under the diaphragm as a

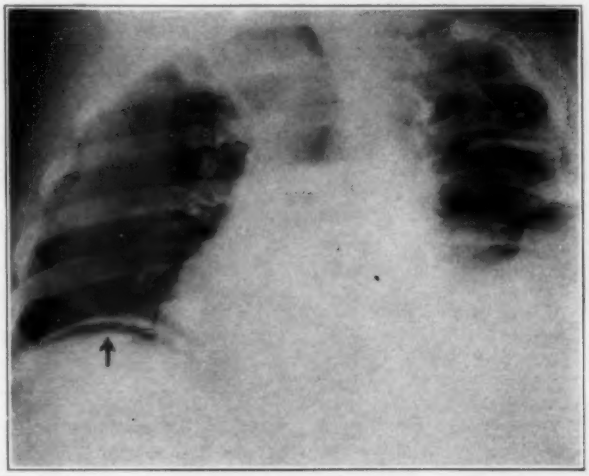

Fig. 1.—An accidental introduction of air under the right cusp of the diaphragm during thoracentesis (the patient in the erect position). The oblique view frequently shows the air to better advantage.

diagnostic procedure, in order to obtain a clearer visualization of the diaphragm or of the dome of the liver. In cases in which a neoplasm or cyst in this region is suspected, a diagnostic insufflation of air in the subphrenic space will reward the examiner with a clear portrayal of the existing pathologic conditions.

As a diagnostic procedure, air or gas is often introduced into the abdomen through the anterior abdominal wall (transabdominal pneumoperitoneum). With the patient in the erect or the semirecumbent position, the films show the accumulation of gas under the diaphragm. Similar findings obtain when air is introduced into the abdomen through the uterus and the patent fallopian tubes (tubal insufflation or Rubin test).

In the majority of cases, air can be demonstrated in the subdiaphragmatic space twenty-four hours or longer after an abdominal laparotomy (fig. 2). The accumulation of air may be very slight but is readily observed in the roentgenograms taken with the patient in the erect position.

ROENTGENOLOGIC APPEARANCE OF CONDITIONS OF AIR

When air between the diaphragm and the liver is free, it has a crescentic appearance, assuming the contour of the diaphragmatic dome above and the arch of the superior surface of the liver below. The air does not appear in the views which are taken with the patient in the prone position. However, when the patient assumes a semirecumbent or an erect posture, the air rises and collects under the diaphragm and so

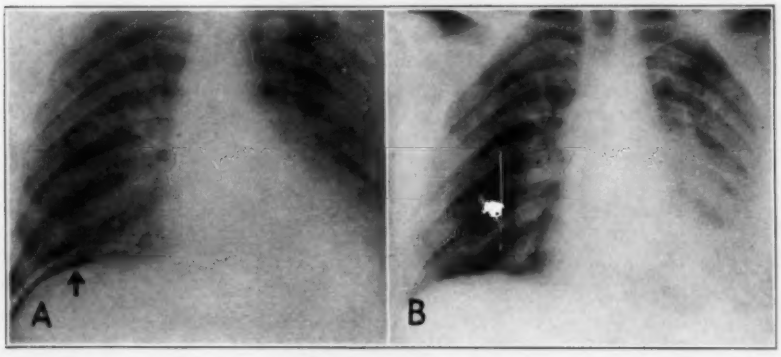

Fig. 2.—*A*, view of the chest of a patient in the erect position, taken three days after appendectomy. The presence of air between the diaphragm and the liver is clearly seen. *B*, view of the chest of the same patient in the horizontal position. The collection of air has disappeared, leaving a normal subdiaphragmatic space.

becomes visible in the film. The gas is sometimes better portrayed in oblique or lateral views.

Not infrequently, a subdiaphragmatic abscess may reveal a horizontal fluid level above which a pocket of gas is present, conforming with the dome of the diaphragm or taking the shape of the upper segment of a circle (fig. 3). This type of infection is caused by an anaerobic or gas-producing organism. The fluid level of the abscess is visualized roentgenologically in erect and lateral decubitus views and is obliterated in views taken with the patient in the prone position. The splash or succussion sound of the fluid and air in the abscess cavity can be elicited roentgenoscopically by shaking the patient. Similar roentgenologic observations prevail in cases of an abscess of the liver which contains gas and pus (fig. 4).

Apparent but not real evidence of the presence of air within the right subphrenic space is observed in patients with situs inversus. In these persons the gas-filled stomach lies in the right upper quadrant of

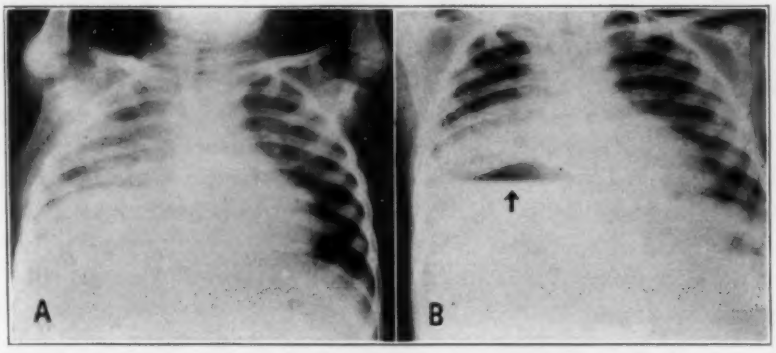

Fig. 3.—Abscess of the right subdiaphragm, showing the elevated cusp and the horizontal fluid level, above which the gas shadow is seen conforming to the dome of the diaphragm. *A*, roentgenogram of the chest of the patient in the prone position. *B*, view of the chest of the patient in the erect position.

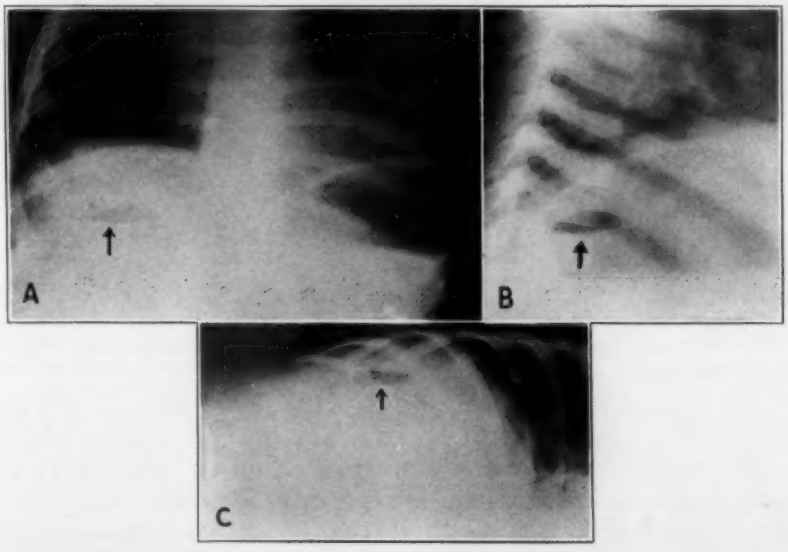

Fig. 4.—Abscess of the liver in a 16 month old infant. *A*, the pocket of gas and the fluid level are visible in the posteroanterior view taken with the patient in the erect position. *B*, view of the chest of the same patient in the upright lateral position. *C*, view of the chest of the same patient in the lateral decubitus position, with the right side up.

the abdomen, immediately beneath the diaphragm, and simulates roentgenologically the appearance of air in the right subdiaphragmatic space. However, an error here is easily avoided by taking note of the right-

sided position of the heart and by observing the passage of an opaque meal through the transposed stomach.

From the clinical aspect, probably the most important cause for the presence of gas beneath the diaphragm is a perforated viscus. Gas escaping from a ruptured peptic ulcer collects in the subdiaphragmatic space when the patient assumes the upright position and is easily recognized on the films (fig. 5). The air disappears from the subphrenic area in the roentgenogram taken with the patient in the prone position. These well known observations have been considered indisputable. Wessler and Jaches [1] described these findings as "such a characteristic feature of the roentgen plate that it may be regarded as positive evidence of free air

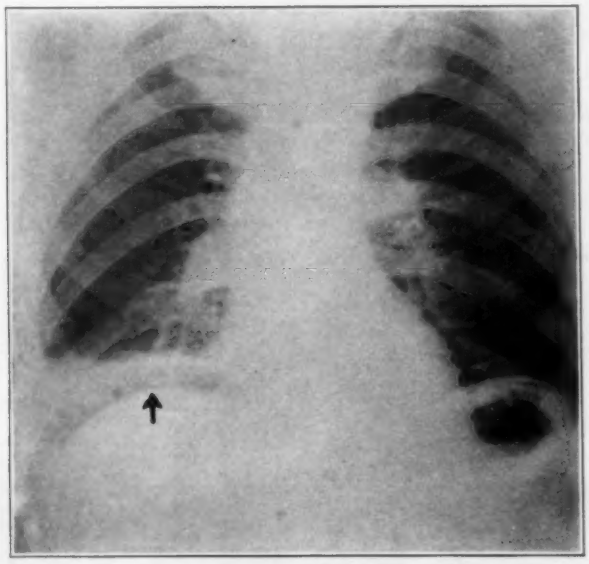

Fig. 5.—A collection of gas under the diaphragm in a patient with perforation of a duodenal ulcer.

or other gas in the peritoneal cavity." Nevertheless, there is another condition which may be readily confused with such a collection of gas and which must be borne in mind in order to avoid serious error.

In the normal person, the hepatic flexure of the colon is found immediately beneath the lower border of the liver. On rare occasions, however, this part of the colon may be found to lie between the diaphragm and the right lobe of the liver. Whether the malposition is congenital or acquired, it is usually observed on flat roentgenograms of the abdomen because of the presence of gas in the interposed segment

1. Wessler, H., and Jaches, L.: Clinical Roentgenology of Diseases of the Chest, Troy, N. Y., The Southworth Company, 1923, p. 509.

of the bowel. The gas-filled hepatic colon is visualized with the patient in the horizontal as well as in the vertical position. One must not confuse this anomalous position of the colon with eventration or herniation of the diaphragm. In hepatodiaphragmatic interposition of the hepatic flexure, the diaphragm is not displaced, and no part of the bowel lies in the chest cavity.

In the presence of this ectopic position of the gas-filled hepatic colon, how can the diagnosis of subdiaphragmatic air be made in a case of perforation of a hollow viscus? The condition of the left subphrenic region is often of little or no help, because of the frequent presence of gas in the stomach and splenic colon, which lie directly underneath the diaphragmatic leaf. The following case report illustrates the difficulty that may be encountered in arriving at the correct interpretation and emphasizes the importance of an exact interpretation of the gas shadow under the diaphragm.

REPORT OF CASES

CASE 1.—F. H., a Negress aged 45, a laundry worker, entered the Jewish Hospital with a history of severe abdominal cramps and vomiting for twenty-four hours. For the past year she had been troubled with "gas" and pyrosis following meals, and these conditions had become aggravated during the past week. Temporary relief was afforded by sodium bicarbonate. On the day prior to hospitalization the patient was suddenly seized with acute cramplike pains in the upper part of the abdomen and occasional vomiting. There was no bloody discoloration of the vomitus and stools. The symptoms persisted through the night and the following morning, at which time she was admitted to the hospital.

Physical examination showed a well developed, well nourished woman. The temperature was 100.4 F., the pulse rate 108 and the blood pressure 110 systolic and 74 diastolic. Except in the abdomen, the findings were normal. The abdomen was definitely rigid, and its lower half appeared distended. The rigidity was slightly more pronounced on the right side than on the left. Generalized rebound tenderness was present, most evident in the upper part of the abdomen and in the right lower quadrant. No masses were felt. Urinalysis gave negative results. The blood count showed 6,000 white blood cells with 66 per cent of polymorphonuclear leukocytes and a marked shift to the left. Plain roentgenograms of the abdomen taken in both the erect and the prone position revealed the manifestations of a pneumoperitoneum, as evidenced in both positions by the collection of a large amount of gas under the diaphragm. The diagnosis of a perforated viscus was ventured.

The patient was immediately operated on. The peritoneal cavity was opened by a right upper rectus incision. Cloudy fluid was obtained, and a pinpoint perforation on the anterior surface of the pylorus was found. Grayish grumous fluid was observed oozing through the opening. The perforation was closed. Peritonitis developed, and the patient died three days later.

Comment.—Although the findings at operation justified the roentgenologic conclusions, a review of the films on the following day raised several disturbing questions. Why was there still a large gas shadow in the subdiaphragmatic space in the views taken with the patient in the

prone position? What was the significance of the thin, dense bands traversing the collection of air? The opinion was expressed that the shadow between the diaphragm and the liver represented not free gas but an interposition of a gas-filled hepatic colon. The traversing thin bands were probably caused by the haustrations of the interposed bowel. The unchanged appearance of the gas shadow in views taken with the patient in both positions, erect and prone, indicated that there was a hepatodiaphragmatic interposition of the hepatic flexure. That free

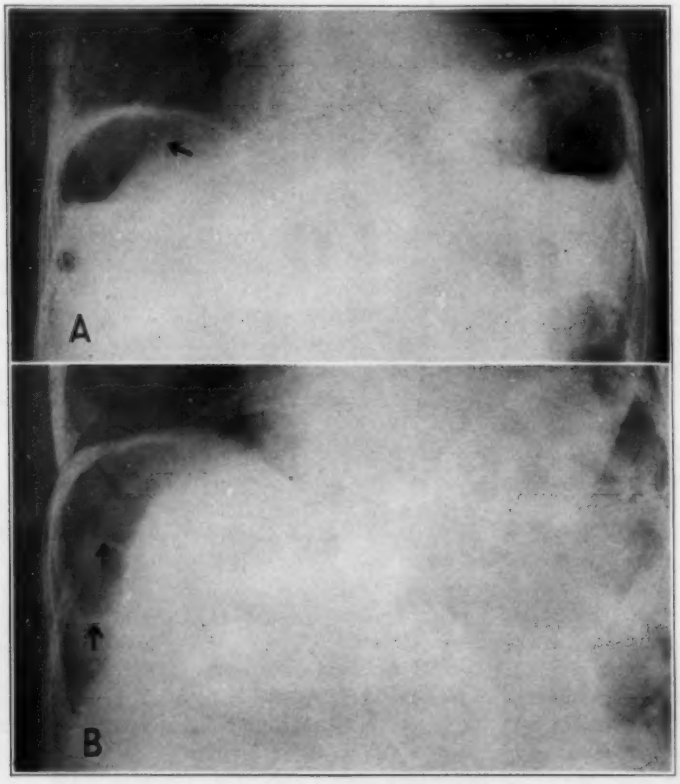

Fig. 6.—*A*, a large collection of subdiaphragmatic gas in a patient with a perforated prepyloric ulcer. Observe the fluid level on the right and the opaque band traversing the air collection (arrow). The roentgenogram was taken with the patient in the erect position. *B*, view of the chest of the same patient in the prone position. A considerable amount of gas is still present between the diaphragm and the liver. The fluid level has disappeared and several thin bands (arrows) are observed. The gas shadow represents a hepatodiaphragmatic interposition of the hepatic flexure, and the traversing bands indicate the haustral shadows. The films show the probable presence of free gas, as evidenced by the fluid level in the erect views in addition to the interposition of the gas-filled colon.

gas was present in the abdominal cavity and rose to the subphrenic space when the patient assumed the erect position was conjectural, since if so it would be obscured by the gas in the interposed bowel. However, the presence of a distinct fluid level in the views taken with the patient in the erect position indicated that, in addition to the meteoric hepatic flexure as revealed by its haustral bands, there were free air and fluid in the subphrenic space. In the roentgenograms taken with the patient in the prone position, with the air and fluid moving away from the diaphragm, the gas-filled bowel was observed unobscured between the liver and the diaphragm. Unfortunately the patient died on the third day after the operation; so further study was not possible. Permission for necropsy was not obtained.

Pendergrass and Kirk [2] reported a case of interposition of the hepatic flexure which was mistaken for free gas in the subdiaphragmatic area. The diagnosis of a ruptured viscus was made. At operation, acute pancreatitis was found. On reviewing the films the authors noticed for the first time the thin haustral bands traversing the gas pocket. Necropsy showed the liver to be rotated and low and the hepatic flexure interposed between the diaphragm and the liver. The case illustrates how an anomalous condition may closely simulate a serious surgical entity.

The following report of the case of a chronically ill patient illustrates a similar abnormal position of the hepatic flexure which had no bearing on the diagnosis.

CASE 2.—A. W., a white man aged 76, was admitted to the Brooklyn Cancer Institute with a history of difficulty in swallowing for four months. This symptom had become progressively worse until at the time of admission he was able to take liquids only. He had lost 45 pounds (20.4 Kg.) in the past four months. Until the onset of this illness, he had enjoyed good health, with no gastric or intestinal complaints.

On physical examination, he appeared well developed and showed evidence of recent loss of weight. The chest was normal except for a few basal rales. Percussion elicited normal diaphragmatic movement. He presented an indefinite mass in the right upper quadrant of the abdomen which did not move with respiration. The abdomen was soft, and normal hepatic dulness was obtained.

During a gastrointestinal roentgen examination a large collection of gas was observed between the diaphragm and the liver (fig. 7). The gas appeared continuous with the rest of the bowel and represented an interposition of the hepatic flexure, which could not be found in its usual position below the liver. Traversing the gas shadow were thin bands representing haustrations of the colon. Even when views were taken with the patient in the reclining position the gas shadow could not be obliterated. The remainder of the roentgenologic study showed the presence of a large filling defect deforming the cardiac end of the stomach and extending to the lower part of the esophagus, which showed a definite constriction, above which the lumen was considerably dilated. The

2. Pendergrass, E. P., and Kirk, E.: Significance of Gas Under the Right Dome of the Diaphragm, Am. J. Roentgenol. **22**:238-246 (Sept.) 1929.

observations were highly suggestive of an advanced gastric malignant process extending to and encroaching on the esophagus.

In successive studies over a period of several weeks, the interposition of the hepatic flexure was not a constant observation. Although this interposition was present on most of the roentgenograms, on several of them the bowel was not shown to be interposed but remained abnormally high. These roentgen observations were clearly visualized with a barium sulfate meal. The right diaphragmatic cusp occupied a normal position and showed free respiratory excursions. Insufflation of the bowel with air (Bastedo), although not entirely satisfactory because of the patient's general weakness and his inability to cooperate, demonstrated an unusually high position of the hepatic flexure (fig. 8).

Comment.—In this case a chronically ill patient presented the clinical and roentgenologic evidence of a carcinoma of the cardia. An inci-

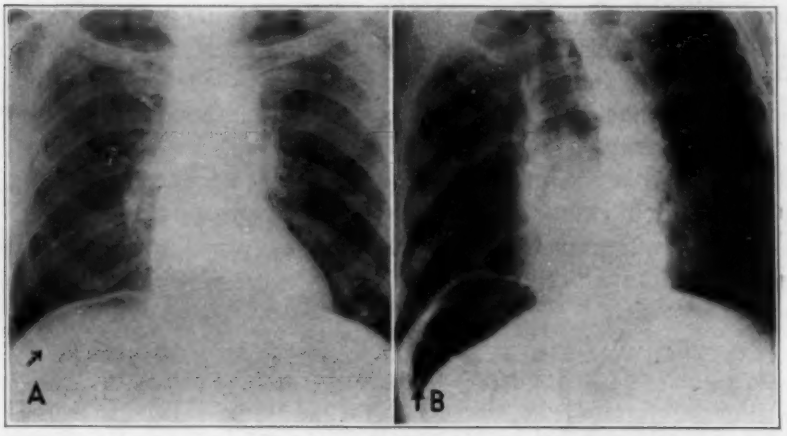

Fig. 7.—*A*, posteroanterior roentgenogram with the patient in the erect posture, showing a trace of gas in the right subphrenic space. Observe the faint shadow of a traversing haustral band (arrow). The view shows an interposition of the hepatic flexure between the liver and the diaphragm. *B*, view of the chest of the same patient in the oblique position. The gas-filled hepatic flexure and its haustral bands (arrows) are best seen in this view.

dental but inconstant observation was the high position of the gas-filled hepatic flexure, which frequently was demonstrated to be interposed between the diaphragm and the liver.

Both case reports present instances of hepatodiaphragmatic interposition of the hepatic flexure. In the first case the interposition tended to obscure the positive roentgenologic evidence necessary to establish the diagnosis of a ruptured viscus, and in the second the anomalous position of the hepatic flexure colon was apparently of no clinical importance. In both cases the roentgen findings were alike, namely, the presence of gas between the diaphragm and the liver, which sometimes

was observed to be continuous with the rest of the bowel, the clear outline of the haustral markings, the absence of the hepatic flexure in its usual position below the liver, and the persistence of the gas shadow in the views taken with the patient in the prone position.

PATHOGENESIS OF INTERPOSITION OF THE HEPATIC FLEXURE

According to Rogers,[3] the first case of hepatodiaphragmatic interposition was reported by Cantani in 1865. Béclère [4] in 1899 described the roentgenologic findings. From that date, sporadic case reports appeared in the records until 1928, when Uspensky [5] recorded 8 cases

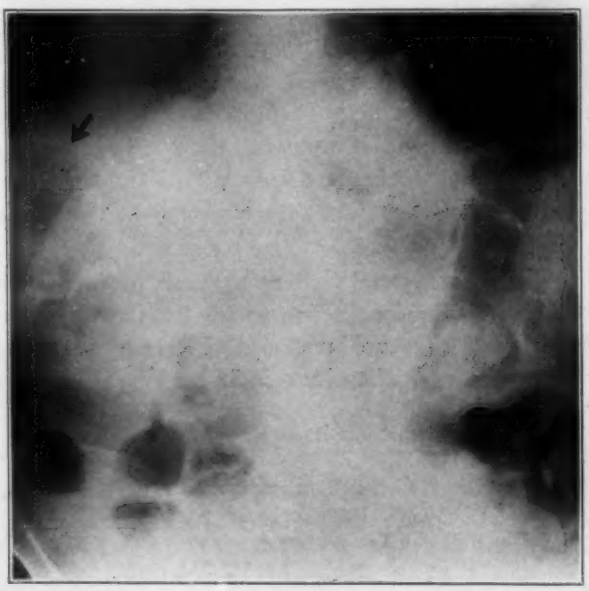

Fig. 8.—View of the chest of the patient six weeks later, after insufflation of the colon with air. The interposition was not permanent. However, the gas shows the unusually high position of the hepatic flexure (arrow).

of permanent and 14 of temporary interposition. Just [6] in 1929 reviewed the literature and presented 3 cases.

3. Rogers, J. C. T.: Hepato-Diaphragmatic Interposition of the Colon, Illinois M. J. **68**:264-268 (Sept.) 1935.

4. Béclère, A.: Rectification d'une erreur de diagnostic: Ectopie du côlon transverse prise, a l'examen radioscopique, pour un abcés gazeux sousphrénique, Bull. et mém. Soc. méd. d. hôp. de Paris **16**:506-507 (May) 1899.

5. Uspensky, A.: Die pathogenetische Bedeutung des Symptomenkomplexes der "Interpositio colonis," Fortschr. a. d. Geb. d. Röentgenstrahlen **37**:540-555, 1928.

6. Just, E.: Zur Frage der Interpositio colonis, Deutsche Ztschr. f. Chir. **220**:334-354, 1929.

The exact causative factors in interposition are unknown. Usually, patients presenting its symptoms show an anomaly in the length of the transverse colon, which is the cause enabling the hepatic flexures to reach such a high position. Other factors are distention of the colon, with rotation on its mesentery, resulting in its elevated position in the abdomen (Vietor [7]), hepatoptosis, with or without shrinkage in the size of the liver and loss of its plasticity (Wessler and Jaches,[1] Assmann,[8] Dehn,[9] and Zucchi [10]), an anomalous development or weakness in the hepatic ligaments (Podkaminsky [11]) and varying degrees of weakness or malfunction of the diaphragm (Slavin [12]). Probably any one of these causative factors or a combination of several may operate in the production of interposition of the rising meteoric colon. When the close contact between the liver and the diaphragm is weakened by any of the causes just mentioned, whether it be paresis of the diaphragmatic cusp due to phrenicectomy on the right side, or ptosis and rotation of the liver as a result of its shrinkage or of defects in its ligamentous attachments, there is a predisposition to hepatodiaphragmatic interposition, provided the length of the colon is sufficient to enable it to rise to this unusual position.

Uspensky,[5] however, stated that as a result of inflammation in the region of the colon adhesions form which fix the bowel in a bizarre position. Soupault,[13] Trémolières and Pierron,[14] Trémolières and Tardieu,[15] Bürger [16] and Weiland [17] emphasized the possibility that some

7. Vietor, A. C.: Anatomic Basis for the Study of Splanchnoptosis, Arch. Surg. **28**:659-683 (April) 1934.

8. Assmann, H.: Die klinische Röntgendiagnostik der inneren Erkrankungen, ed. 4, Leipzig, F. C. W. Vogel, 1929, p. 788.

9. Dehn, O.: Ein Fall von Lungentumor mit ungewöhnlichem Röntgenbefund, Fortschr. a. d. Geb. d. Röntgenstrahlen **34**:333-334, 1926.

10. Zucchi, L.: L'interposizione epatodiaframmatica del colon, Riforma med. **49**:882-888, 1933.

11. Podkaminsky, N. A.: Zur Frage nach den Ursachen der Interposition von Organen zwischen Diaphragma und Leber, Fortschr. a. d. Geb. d. Röntgenstrahlen **36**:327-333, 1927.

12. Slavin, P.: Interposition of the Colon Following Induced Phrenic Paralysis, Am. J. Roentgenol. **33**:481-485 (April) 1935.

13. Soupault, R.: Interposition inter-hépato-diaphragmatique du côlon. Diverticule du duodénum. Ulcus térébrant du duodénum, Arch. d. mal. de l'app. digestif **20**:350-355 (March) 1930.

14. Trémolières, F., and Pierron, E. J. M.: L'interposition hépato-diaphragmatique du côlon, Presse méd. **38**:1-3 (Jan. 1) 1930.

15. Trémolières, F., and Tardieu, A.: L'interposition hepato-diaphragmatique du côlon, Arch. d. mal. de l'app. digestif **21**:1154-1197 (Dec.) 1931.

16. Bürger, M.: Zur Klinik der Leberdystopien, Klin. Wchnschr. **4**:102-107, 1925.

17. Weiland, W.: Ein röntgenologisches Phänomen bei perforiertem Magengeschwür, München. med. Wchnschr. **62**:537-538 (April) 1915.

type of inflammatory process, such as a perforating duodenal or pyloric ulcer, may result in the formation of pericolic adhesions which fix the hepatic flexure in an abnormal site. According to Podkaminsky,[11] abnormal intra-abdominal pressure tends to weaken the contact between the liver and the diaphragm and to produce interposition.

SYMPTOMS AND TREATMENT

Often there are no clinical symptoms, and the condition may be found accidentally during a roentgenographic examination for other reasons (case 2). However, not infrequently these patients are of the visceroptotic type and complain of localized or generalized abdominal pain, which may radiate to the right shoulder or be referred to the abdomen and which is aggravated by deep inspiration. Abdominal distention and constipation are often present. Anterior hepatic dulness may be lost, and a mass formed by the displaced liver may be palpable in the right lower quadrant of the abdomen. The clinical picture is frequently overshadowed by some other organic lesion, such as cholecystitis, peptic ulcer or intestinal obstruction. Roentgenologic examination establishes the diagnosis.

If the symptoms are mild, conservative measures are employed to relieve the discomfort caused by meteorism and constipation. However, when a permanent incarceration of the bowel produces severe symptoms, surgical intervention is suggested, and some form of hepatopexy is performed.

SUMMARY

The causes for accumulation of gas in the right subdiaphragmatic space are enumerated, and their recognition in the roentgenograms is described. Two cases of hepatodiaphragmatic interposition of the hepatic flexure are reported. In the first case this lesion was confused with gas in the subphrenic space from a ruptured peptic ulcer; in the second it appeared as an accidental finding. Finally, a brief résumé is given of the history and pathogenesis of the causative factors in the production of interposition, and mention is made of the possible symptoms and management of this anomalous condition.

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MECHANICS OF SIMPLE INTESTINAL OBSTRUCTION

AN EXPERIMENTAL STUDY

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An enormous amount of research has yielded considerable information about intestinal obstruction and has resulted in the modification of previous concepts. Admitting that progress has been made, it is evident from a perusal of recent figures on mortality that much remains to be learned. Christopher,[1] Miller [2] and others have recently reported mortality rates varying between 40 and 60 per cent. When a disease carries such a mortality, no apology need be made for presenting further experimental studies. The fact that over 5,000 articles dealing with intestinal obstruction are on file in the library of the Surgeon General is an indication of the interest of physicians in this disease and an admission that their knowledge of the subject is far from complete.

It is evident that when complete knowledge of the physiologic changes that occur in intestinal obstruction is acquired, physicians will be better equipped to cope with the problem. Any alteration of normal physiology which takes place must be secondary to the stasis which is the immediate effect of intestinal obstruction. Stasis leads to distention and to an increase of intraenteric pressure, and these are the primary causes of the complications which appear when obstruction occurs. The effect of distention on the wall of the bowel is best stated in terms of the effect of various degrees of increased intraluminary pressure. This work deals with the subject of increased intraenteric pressure as it occurs in simple ileal obstruction and the effects of such pressure on the structure, the function and the permeability of the intestinal wall.

This presentation is concerned only with a study of the mechanism of simple obstruction of the ileum. The problem of strangulation obstruction is not of concern here, except as features of strangulation may attend increased intraenteric pressure. A consideration of the

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Abridgment of thesis submitted to the Faculty of the Graduate School, University of Minnesota, in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Surgery.

1. Christopher, F., and Jennings, W. K.: Mortality in Intestinal Obstruction, Ann. Surg. **99**:332-337, 1934.

2. Miller, C. J.: A Study of Three Hundred and Forty-Three Surgical Cases of Intestinal Obstruction, Ann. Surg. **89**:91-107, 1929.

various clinical types of obstruction also lies outside the province of this paper. The work is confined to a study of ileal obstruction for two reasons: First, there is a comparative dearth of experimental observation on this type of obstruction as compared with an abundance of material on high intestinal obstruction. Second, the cause of death following low ileal obstruction is still obscure, while the lethal factors in simple high intestinal obstruction are fairly well understood. Whenever possible, an effort has been made to compare the effects of various experimental conditions on the normal intestinal wall with their effects on the wall of the obstructed intestine.

INTRAENTERIC PRESSURE IN INTESTINAL OBSTRUCTION

In order to determine the effect of increased intraenteric pressure on the intestinal wall it is necessary first to know what degree of pressure is present in experimental and in clinical cases of intestinal obstruction.

The normal sustained intraintestinal pressure in dogs is 2 to 4 cm. of water (Owings and others [3]). In simple obstruction of the ileum the sustained pressure may be increased to 8 cm. of water. In closed jejunal loops Burget [4] has recorded pressures as high as 50 or 60 cm. of water. It is evident that the pressure reached a higher level in a shorter period in closed ileal loops than in simple obstruction, in which the bowel may empty itself by reversed peristalsis and regurgitation.

Stone and Firor [5] recorded a pressure of 150 cm. of water in a patient with low ileal obstruction of several hours' duration. As far as can be determined, this is the only clinical measurement, besides my own, of the pressure in intestinal obstruction in man. Elman and Aird [6] measured the intraenteric pressure in a human being with a jejunal fistula. A sustained pressure varying from 2 to 4 cm. of water was noted. During peristalsis the pressure rose rapidly, reaching 15 to 27.5 cm. of water. Minor peristaltic waves up to 6 or 7 cm. of water were frequent. With increased peristalsis there was always associated mild hypogastric discomfort. Colic was present when the pressure

3. Owings, J. C.; McIntosh, C. A.; Stone, H. B., and Weinberg, J. A.: Intra-Intestinal Pressure in Obstruction, Arch. Surg. **17**:507-520 (Sept.) 1928.

4. Burget, G. E.; Martzloff, K.; Suckow, G., and Thornton, R. C. B.: Closed Intestinal Loop: I. Relation of the Intraloop (Jejunum) Pressure to the Clinical Condition of Animal, Arch. Surg. **21**:829-837 (Nov.) 1930.

5. Stone, H. B., and Firor, W. M.: Absorption in Intestinal Obstruction: Intra-Intestinal Pressure as a Factor, Tr. South. S. A. **37**:173-184, 1924.

6. Elman, R., and Aird, I.: Observations on Intra-Intestinal Pressure with Reference to Absorption of Saline, Proc. Soc. Exper. Biol. & Med. **32**:1620-1622, 1935.

reached 20 cm. of water. On one occasion, after the intake of food, a pressure of 35 cm. of water was noted.

The intraenteric pressure was determined in 25 dogs with low ileal obstruction of one to seven days' duration.[7] The pressure ranged from 4 to 19 cm. of water, or roughly four times the normal pressure. Active peristalsis was uniformly present even after seven days of obstruction (fig. 1). The intraenteric pressure in this series apparently increased with the duration of the obstruction. It was observed that the sustained pressure in low ileal obstruction was seldom above 14 cm. of water (table 1). These data are in accord with the findings of Owings and his associates.[3]

Opportunity has also been afforded to determine the intraenteric pressures at the time of the operation in 6 clinical cases of mechanical

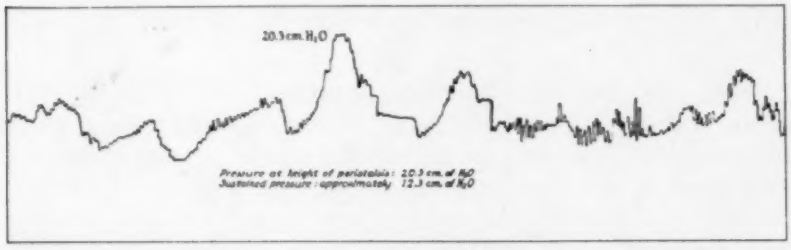

Fig. 1.—Intraenteric pressure and peristalsis in simple ileal obstruction of seven days' duration. (Pressure at height of peristalsis, 20.3 cm. of water; sustained pressure, approximately 12.3 cm. of water.)

TABLE 1.—*Sustained Intraenteric Pressure in Dogs with Simple Ileal Obstruction*

Dog	Duration of Obstruction, Days	Pressure, Cm. H_2O	Dog	Duration of Obstruction, Days	Pressure, Cm. H_2O
1	1	8	11	4	14
2	2	4	12	4	10
3	2	6	13	4	7
4	2	12	14	4	12
5	2	6	15	4	10
6	3	10	16	4	8
7	3	12	17	4	10
8	4	8	18	7	19
9	4	10	19	7	12
10	4	14			

obstruction of the small bowel.[8] The sustained pressure varied between 4 and 18 cm. of water. With peristaltic activity, pressures rose to 20 and then to 30 cm. of water.

7. Sperling, L.; Paine, J. R., and Wangensteen, O. H.: Intra-Enteric Pressure in Experimental and Clinical Intestinal Obstruction, Proc. Soc. Exper. Biol. & Med. **32**:1504-1506, 1935.

8. Sperling, L.: Role of the Ileo-Cecal Sphincter in Cases of Obstruction of the Large Bowel, Arch. Surg. **32**:22-49 (Jan.) 1936.

Pressures were observed at operation when colostomy was performed for the relief of distention in 8 cases of acute obstruction of the colon. The sustained pressure varied between 12 and 52 cm. of water; in 6 cases pressures above 23 cm. of water were recorded, much higher pressures than were observed in cases of obstruction of the small bowel. The significantly greater pressure in obstruction of the colon is due to the action of the ileocecal sphincter, which makes the colon virtually a closed loop, in which the intraenteric pressure usually greatly exceeds pressures observed in simple ileus. These observations have been reported at greater length elsewhere.[8]

INFLUENCE OF SIMPLE OBSTRUCTION ON THE PHYSICAL CHARACTER OF THE INTESTINAL WALL

There is relatively little in the literature concerning the changes in the physical character of the wall of the bowel following simple obstruction. In the following sections, a detailed study is presented of the influence of simple mechanical ileus on the length, the weight, the water content, the blood content, and the strength of the intestinal wall.

INFLUENCE OF OBSTRUCTION ON THE LENGTH OF THE BOWEL

According to several investigators the first response of the bowel of the guinea pig to distention is a contraction of the longitudinal muscles (Trendelenburg,[9] Crane and Henderson[10]). This produces a decrease in length which amounts to as much as 20 or 25 per cent of the entire length of the bowel. Van der Reis and Schembra[11] presented observations to show that the functional length of the small bowel is only 2.2 to 2.7 meters, or 6 to 15 feet. They contended that in the living person the bowel is much shorter than in the cadaver, in which it usually measures about 22 feet (6.6 meters). In instances of fistula in the terminal ileum, a catheter 6 to 9 feet (1.8 to 2.7 meters) long was found to suffice for the entire length of the small bowel.

No definite information is available as to the effect of obstruction on the length of the bowel. Because of the difference of opinion among clinicians concerning this matter, it was felt worth while to investigate it. It was found that in experimental simple obstruction of the ileum

9. Trendelenburg, P.: Physiologische und pharmakologische Versuche über die Peristaltik des Dünndarms, Arch. f. exper. Path. u. Pharmakol. **81**:55-129, 1916-1917.

10. Crane, J. W., and Henderson, V. E.: The Sensitivity of the Small Intestine at Different Levels to Internal Pressure, Am. J. Physiol. **70**:22-25, 1924.

11. van der Reis and Schembra, F. W.: Weitere Studien über die funktionelle Darmlänge; operative Ergebnisse und Beobachtungen an Bauchfenster, Ztschr. f. d. ges. exper. Med. **52**:74-83, 1926.

there was a definite shortening of the intestine (Sperling and Wangensteen [12]). The length of a segment which before obstruction measured 12 inches (30 cm.) decreased by as much as one third after obstruction. Statistically, it was shown that the intestine is shortened to a significant degree in simple ileal obstruction. Hypertonicity of the obstructed intestine, with marked contraction of the longitudinal muscles, is probably responsible for this shortening. The shortening is sufficient to account for some of the increased weight of the wall of the bowel which is observed in simple ileal obstruction.

TABLE 2.—*Shortening of the Bowel in Simple Ileal Obstruction—Ultimate Length of Consecutive 12 Inch Segments from Site of Obstruction, Ileum to Jejunum*

Dog	Duration of Obstruction, Days	Segments 1	2	3	4	5	6	7	8	9
1	4	8.75	9.00	7.50	9.00	....	....	....	....	9.50
2	4	12.00	12.00	8.50	9.50	9.50	....	....	....	9.50
3	4	9.50	9.00	8.00	7.00	....	....	....	....	....
4	4	8.25	7.00	7.25	7.25	....	....	....	....	7.75
5	4	7.25	6.75	7.00	7.00	8.25	8.50	....	....	8.25
6	4	9.25	8.75	8.50	13.50	6.75	....	....	....	7.25
7	2	9.50	10.00	10.00	11.50	10.50	12.00	11.00	....	9.00
8	2	8.75	8.25	7.75	8.75	7.75	8.50	9.25	11.00	10.00
9	1	10.50	10.50	(Only two lower segments marked off)						
10	1	8.50	5.50	(Only two lower segments marked off)						
11	3	10.00	8.00	(Only two lower segments marked off)						
12	3	8.50	9.75	(Only two lower segments marked off)						

Average shortening: -2.77 ± 0.35 inches; -3.47 ± 0.40 inches

TABLE 3.—*Length of Consecutive 12 Inch Segments of the Small Intestine of Normal Dogs Measured After Forty-Eight Hours (Ileum to Jejunum)*

Dog 1.....	12.00	11.25	12.5	13.00	9.5	12.00	12.50	13.5	13.00	11.00	
Dog 2.....	11.25	13.25	11.0	11.25	11.0	10.25	10.25	12.0	10.75	10.25	10.25	11.0

Average decrease in length of 0.63 ± 0.23 inch

INFLUENCE OF OBSTRUCTION ON THE WEIGHT OF THE INTESTINAL WALL AND ON ITS CONTENT OF WATER AND HEMOGLOBIN

As far as can be determined, there has been no previous estimation of changes in the weight of the intestine in simple intestinal obstruction. Opportunity to make such observations was afforded in the course of other experiments on dogs with simple obstruction of the ileum.

12. Sperling, L., and Wangensteen, O. H.: Influence of Obstruction of the Bowel upon Its Length and Weight, Proc. Soc. Exper. Biol. & Med. **32**:1219-1224, 1935.

The weight of the ileum immediately above the site of obstruction was determined to be 114 per cent greater than that of the normal ileum (Sperling and Wangensteen [12]). When the factor of shortening of the bowel was eliminated, the true gain in weight was found to be 34 per cent (table 6). Comparisons of the water content of the normal and that of the obstructed bowel showed an average increase of about 7 per cent in the ileum above the site of obstruction and an average

TABLE 4.—*Weight of Consecutive Feet of Normal Intestine from the Ligament of Treitz to the Terminal Portion of Ileum*

Dog	Weight, Kg.	Weight of Consecutive 1 Foot Segments of Bowel, Jejunum to Ileum, Gm.
1	16	47—40—40—45—43—43—39
2	21	36—38—40—40—37—29—26
3	40	46—44—42—42—47—44—40—36
4	22	39—35—32—32—30—25—26—24—20—25—24
5	25	42—40—40—39—36—38—35—35—34
6	8	37—33—30—32—26—18
7	14	34—33—32—32—30—26—22—20—23—21
8	17	34—27—27—28—30—28
9	18	45—39—37—33—29—30—29
10	22	40—34—27—32—33—30—29
Average weight.....	20	Av. wt. of 1 foot of terminal ileum, 28.7 Gm.

TABLE 5.—*Weight of Consecutive Feet of Obstructed Intestine from the Ligament of Treitz to the Site of Obstruction in the Terminal Portion of the Ileum*

Dog	Weight, Kg.	Duration of Obstruction, Days	Weight of Consecutive 1 Foot Segments of Bowel, Jejunum to Ileum, Gm.
1	18	7	42—45—51—52—62—70—60—85
2	19	8	41—34—36—36—42—40—41—41—49—68
3	19	6	52—45—40—54—50—43—62
4	19	5	46—44—36—50—55
5	28	6	39—43—48—42—48—62
6	17	4	43—52—50—39—46—55
7	16	6	42—46—46—54
8	18	6	39—39—43—56
9	12	7	30—34—49—52—48
10	15	5	56—56—58—72
11	19	7	29—21—20—19—23—26—30—51
Average weight.....	17		Av. wt. of terminal ileum per ft., 60.7 Gm. Percentage of increase, 114

increase of 4.7 per cent in the jejunum remote from the obstruction (table 7). There is evidently no dehydration of the wall of the bowel even though the systemic tissues are desiccated and the blood is concentrated.

The weight per foot of the normal small intestine usually decreases from the duodenum to the terminal portion of the ileum. There seems to be a downward gradient even in weight. In simple ileal obstruction there is a reversal of the normal gradient in weight; that is, weight of the ileum is greatest above the site of obstruction and there is a

decrease in weight in a retrograde manner from the obstructed ileum to the duodenum. This is due to the fact that the segments of bowel immediately above the obstruction increase most in weight while those segments of the jejunum remote from the site of obstruction increase only slightly in weight.

The results indicated in table 8 represent the relative increase in hemoglobin content of the obstructed bowel as compared with that of the

TABLE 6.—*Weight Changes of the Bowel in Simple Ileal Obstruction (1 Foot Segments Marked Off at the Time Obstruction Was Made)*

Dog	Weight, Kg.	Duration of Obstruction, Days	Weight of Consecutive 1 Foot Segments of Bowel, Jejunum to Ileum, Gm.
1	14	4	40—32—31—26—29—52—41
2	13	4	21—22—23—30—34
3	14	4	41—45—44—42—57—58
4	18	4	27—25—35—33—37—35
5	8	4	23—16—18—19—35
6	17	4	31—31—35—50
7	10	4	24—20—21—21—27—27—27—39
8	11	2	22—22—20—19—22—23—22—26
9	13	2	25—24—22—25—23—23—24—26—30
10	14	3	21—20—22—22—29—37
Average weight	13		Average weight of terminal portion of ileum per foot.....38.5 Gm. Percentage of increase.....34

TABLE 7.—*Water Content of Normal Bowel as Compared with Obstructed Bowel*

Normal Bowel			Obstructed Bowel		
Dog	Jejunum, %	Ileum, %	Dog	Jejunum, %	Ileum, %
1.....	76.5	72.6	1.....	82.4	85.1
2.....	79.6	78.1	2.....	84.7	85.0
3.....	76.3	78.2	3.....	84.1	80.5
4.....	77.2	77.2	4.....	78.0	80.9
5.....	78.1	77.7	5.....	77.4	83.3
6.....	76.2	75.5	6.....	83.3	85.4
7.....	75.9	77.8	7.....	81.2	81.2
8.....	70.2	72.7	8.....	81.0	78.4
9.....	77.4	77.5	9.....	80.4	83.5
Average.....	76.3	76.5	Average.....	81.0	83.5
Mean difference	±1.7	±1.73	Mean difference	±2.39	±2.02

In simple ileal obstruction there is approximately 4.7 per cent increase of the water content of the jejunum and 7 per cent increase of the water content of the ileum.

normal bowel (experiment performed on the same dog prior to obstruction). There is a wide range of individual variation. It is noteworthy that an increase of more than 100 per cent is the rule. A previous study made in this laboratory by Carlson and Wangensteen [13] indicated that the accumulation of blood in the bowel in simple obstruction is due to stasis in the vessels and not to interstitial hemorrhage.

13. Carlson, H. A., and Wangensteen, O. H.: Histologic Study of the Intestine in Simple Obstruction, Proc. Soc. Exper. Biol. & Med. **29**:421-424, 1932.

These observations may be briefly summarized as follows:

1. The wall of the bowel above the site of a simple ileal obstruction increases more than 100 per cent in weight.

2. When the factor of shortening of the bowel is eliminated, there is an actual increase in weight of 34 per cent.

3. Edema of the intestinal wall accounts for only 7 per cent of the increase in weight.

4. The increase of blood in the wall of the bowel is the most important factor in the actual increase in its weight.

INFLUENCE OF OBSTRUCTION OF THE BOWEL ON ITS TENSILE STRENGTH (BURSTING STRENGTH)

The changes in the bowel in intestinal obstruction due to increased intraenteric pressure have been emphasized by many investigators.

TABLE 8.—*Comparative Increase of Hemoglobin in the Blood of the Bowel Wall in Simple Obstruction*

Dog	Duration of Obstruction, Days	Comparative Increase, Times Normal
1	1	5.34
2	1	2.76
3	2	3.22
4	2	2.80
5	3	1.40
6	3	1.20
7	4	10.65
8	4	7.17
9	4	2.75
10	4	4.54

Perforations of the bowel due to gangrene are sometimes terminal complications of simple intestinal obstruction.

A study was made to compare the bursting pressure of the wall of the normal bowel with that of the bowel in simple intestinal obstruction (Sperling and Wangensteen [14]). The normal ileum of the dog in most cases was found able to withstand greater degrees of pressure than could the jejunum. The ileum resisted pressures varying between 300 and 1,000 mm. of mercury. The bursting pressure of the jejunum fell within the same limits, but in almost every case was lower than that of the ileum in the same dog. This fact, however, could not be proved to be statistically significant.

In the dogs with intestinal obstruction the reverse was found to be true. The jejunum, that is, that portion of the bowel farthest away from and least affected by the obstruction, was the more resistant.

14. Sperling, L., and Wangensteen, O. H.: Influence of Obstruction of the Bowel on Its Strength (Bursting Strength), Proc. Soc. Exper. Biol. & Med. **32**:1183-1185, 1935.

The tensile strength of the jejunum approached the normal, whereas that of the obstructed segment of bowel (ileum) was far below normal. In the obstructed specimens (ileum) the serosa usually split at the antimesenteric border at a pressure of about 100 to 300 mm. of mercury and soon burst, whereas in the specimens of normal ileum the serosa was usually able to resist a pressure of more than 400 mm. of mercury.

TABLE 9.—*Tensile Strength (Bursting Pressure) of Normal Bowel* *

Dog	Pressure (Mm. Hg) at Which Serosa Split		Pressure (Mm. Hg) at Which Bowel Burst	
	Ileum	Jejunum	Ileum	Jejunum
1	360 a	210 a	620	400
2	300 a	660 a	560	660
3	540	400 p	540	400
4	480 a	440	480	440
5	660 a	600 a	Ends blew out	
6	540 a	610 a	Ends blew out	
7	700 a	540 a	700	540
8	1,060 a	820 a	1,060	820
9	560 p	640 p	560	640
10	700 p	780 a	700	780
11	300 m	220 m	660 m	500 a
12	1,080 m	400 a	1,080	820
Mean	606.7	526.7	696	600

* In this table a indicates antimesenteric border; p, paramesenteric border, and m, mesenteric border.

TABLE 10.—*Tensile Strength (Bursting Pressure) of Obstructed Bowel* *

Dog	Duration of Obstruction, Days	Pressure (Mm. Hg) at Which Serosa Split		Pressure (Mm. Hg) at Which Bowel Burst	
		Ileum	Jejunum	Ileum	Jejunum
1	6	195 a	...	200-End b. o.	
2	6	220 a	240	230	540
3	7	200	200	280	225
4	7	90 a	190 a	180 a	260 a
5	7	200	200	200	200
6	7	200 a	500 a	280	760
7	5	210 a	420 a	250 m	620 m
8	5	360 a	560 p	480 m	560 p
9	5	260 a	360 a	360 a	400 a
10	6	400 m	500	460 m	520 a
Mean		233.51	352.2	292.0	458.9

* In this table a indicates antimesenteric border; p, paramesenteric border, and m, mesenteric border.

These observations appear to justify the following conclusions:

1. The intestinal wall in simple obstruction cannot withstand as high an intraenteric pressure as can the wall of the normal bowel.

2. The portion of the bowel just above the site of obstruction is most affected.

3. The most frequent site of tearing of the serosa and of rupture is the antimesenteric border of the bowel.

INFLUENCE OF SIMPLE INTESTINAL OBSTRUCTION AND OF INCREASED INTRAENTERIC PRESSURE ON THE FUNCTION OF THE BOWEL

Much evidence has accumulated to show that distention, with its increased intraenteric pressure has a pernicious effect on the function of the bowel. Distention produces an alteration in normal physiology which is sufficient to explain many of the findings in cases of intestinal obstruction.

EFFECT OF DISTENTION ON INTESTINAL TONE AND PERISTALSIS

It is recognized that a certain degree of intraenteric pressure acts as a stimulus to peristaltic activity. Ganter [15] and others have determined that a certain critical pressure must be reached in the bowel before peristalsis begins. Below this pressure, which is estimated to be 15 cm. of water (Ganter) for man, peristalsis ceases. This pressure varies from person to person but remains constant in the same person at all times. The normal frequency of peristalsis in the duodenum of man is from 10 to 12 waves per minute. If the pressure is maintained above the critical level, regular rhythmic contractions set in. There is no increase in rate but there is a definite increase in the height of the individual contractions.

Unstriated muscle responds to an increased load (stretch stimulus) with a more powerful contraction. The load in intestinal obstruction is the distention incident to stasis. If peristalsis is absent, it is induced by distention. Bayliss and Starling [16] in 1899 showed that distention resulted first in slight augmentation of the contractions of the intestine, and that prolonged distention produced a strong tonic contraction with rhythmic intermissions. In 1923 Bauer [17] showed that sustained elevated intraenteric pressure resulted at first in increased peristalsis, which disappeared with fatigue. After a short period, peristalsis reappeared, to complete a cycle of alternate activity and rest. I have made the same observation in loops of the intestines of dogs subjected to pressures varying between 20 and 100 cm. of water for a period of one hour. This phenomenon corresponds well to the intermittent cramplike pains suffered by patients with mechanical intestinal obstruction.

Critical Pressure.—The pressure within closed ileal loops was gradually increased by filling the lumen with water at a temperature of 35 to 40 C. Peristalsis

15. Ganter, G.: Experimentelle Untersuchungen über die Peristaltik des menschlichen Dünndarmes, Arch. f. d. ges. Physiol. **201**:101-116, 1923.

16. Bayliss, W. M., and Starling, E. H.: The Movement and Innervation of the Small Intestine, J. Physiol. **24**:99-143, 1899.

17. Bauer, M.: Studien über die Dünndarm Peristaltik, Arch. f. exper. Path. u. Pharmakol. **100**:93-111, 1923.

was absent at pressures below 10 mm. of mercury. It usually began at pressures varying between 10 and 15 mm., the critical pressure for that segment of bowel (fig. 2).

With increasing distention the peristaltic waves were usually augmented in size until a pressure varying between 15 and 30 mm. of mercury was reached.

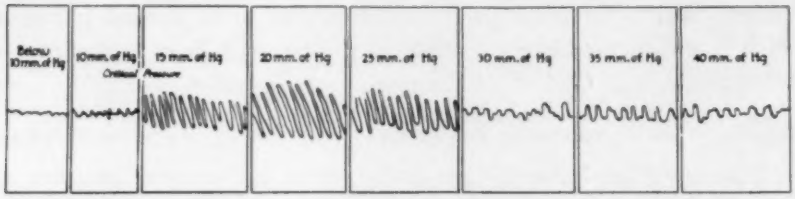

Fig. 2.—Effect of increased intraenteric pressure on peristalsis of the ileum.

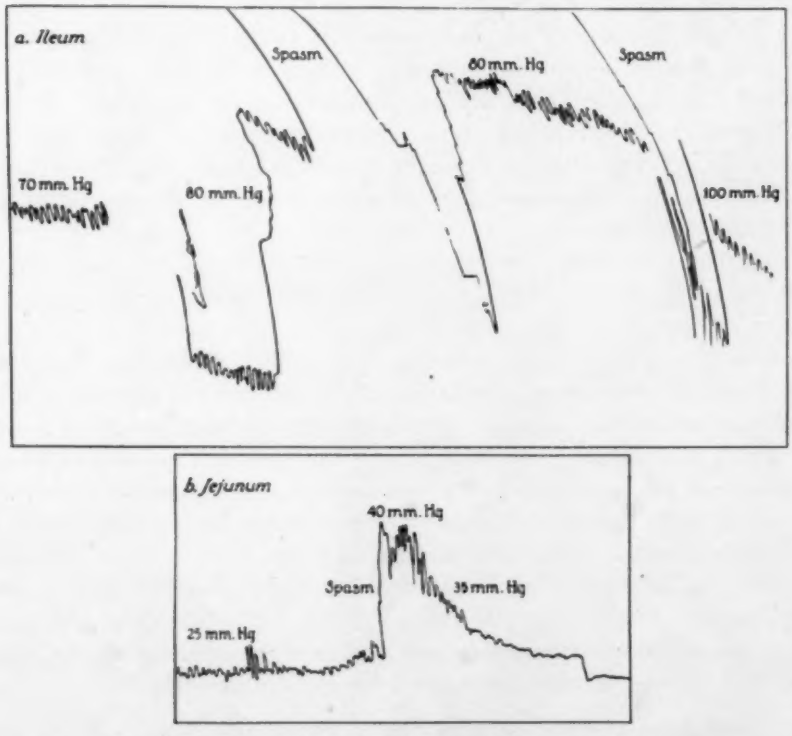

Fig. 3.—Spasm induced by increased intraenteric pressure.

After a pressure of 30 mm. of mercury was reached there was no further increase in the size of the peristaltic waves.

When intestinal loops were subjected to pressures varying between 40 and 80 mm. of mercury for sixty minutes or more, spasmodic contractions of the bowel similar to those occurring in the clinical subjects, that is, to intermittent colic, were occasionally seen (fig. 3). This, however, was not a constant finding. With the animal under light anesthesia, these contractions were accompanied by evidence of pain.

In a jejunal loop, such spasmodic contraction of the bowel was produced with a much lower pressure, that is, 25 mm. of mercury for one hour. During the violent contractions of the bowel, the pressure within the lumen was raised from 25 to 40 mm. of mercury (fig. 3).

To determine the optimal intraenteric pressure for peristalsis, the pressure in the loop of jejunum was gradually increased to 45 mm. of mercury by injecting fluid into the lumen of the bowel. When the tube was clamped, the pressure gradually decreased as the walls of the bowel relaxed. It was noted that with the decrease in pressure peristalsis was at first increased, reaching a maximum at a pressure of 20 mm. of mercury, and then decreased with further diminution in pressure.

Effect of Sudden Decompression of the Bowel on Peristalsis.—When a loop of bowel which had been subjected to increased intraenteric pressure was suddenly decompressed, it responded with violent peristalsis (fig. 4). Loops of ileum subjected to a pressure varying between 40 and 100 mm. of mercury for thirty to sixty minutes responded with violent peristalsis when suddenly emptied. One of these loops very rapidly built its pressure up to 40 mm. of mercury and showed violent peristaltic waves. The rapid increase in tone and augmentation of peristalsis

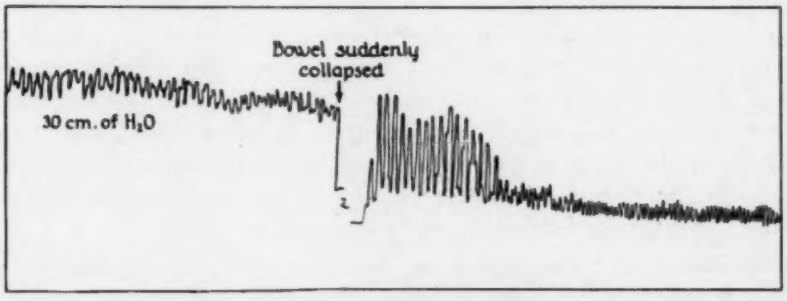

Fig. 4.—Effect of sudden decompression of the ileum on peristalsis.

are the usual reaction of the normal viable bowel to decompression. It is conceivable that if the distention of the intestine were maintained long enough to interfere with its viability the bowel would not respond in this manner. This was noted when pathologic changes (necrosis) occurred owing to prolonged distention. When a loop of ileum which had been subjected to simple obstruction for forty-eight hours was distended to a pressure of 60 mm. of mercury for one hour and then suddenly decompressed, no increased peristalsis was noted.

Relation of Postural Tone to Distention.—It is well known that when hollow viscera are distended the walls of these organs relax and the pressure is gradually reduced. Sherrington [18] has referred to this phenomenon as "postural tone."

The tone of the intestinal musculature is apparently more important than the volume of the intestinal content in affecting peristalsis and intraenteric pressure. The increased pressure is dependent to a greater

18. Sherrington, C. S.: Postural Activity of Muscle and Nerve, Brain **38**:191-234, 1915.

degree on the response of the intestinal musculature to the distention incident to stasis than on simple increase of content. It was noted that at a pressure of 25 mm. of mercury, with active peristalsis in the jejunum, aspiration of 10 cc. from the ileal loop had no effect on the pressure. Apparently changes in tone make up for small changes in volume. This conforms with the law of postural tone as postulated by Sherrington. Massive reduction of volume, that is, sudden decompression, results, however, in a lowering of the pressure. This is accompanied by a great increase in tone, as evidenced by the violent peristalsis and the consequent rapid increase of intraluminary pressure when distended loops of bowel are rapidly decompressed.

No doubt some degree of protection is afforded the wall of the bowel by its ability to dilate and to accommodate itself to increased tension. There is a progressive increase in its diameter in proportion to the duration of the obstruction; that is, the diameter of bowel obstructed for a period of ninety-six hours is greater than that of bowel obstructed for only twenty-four hours. This increase is due in part to stretching caused by the increased intraenteric pressure, but it is principally due to relaxation of the intestinal musculature. The tension on any part of the intestine is dependent on its circumference and on the intraenteric pressure. It is well known that after complete obstruction of the left half of the colon perforation of the bowel is observed most frequently in the cecum. This may be due to the fact that, having the greatest lumen, it is subjected to the greatest pressure.

Sustained high pressure is more damaging to the bowel than intermittent pressure, even if the latter is of so high a degree as to cause local blanching. Ample opportunity is afforded the bowel to dilate and to return to its normal state in the interval between contractions. This pressure-free interval is lacking, of course, when the intraenteric pressure is sustained at a constant level. The insignificant histologic changes observed in experimental and clinical cases of simple intestinal obstruction may be accounted for by this ability of the bowel to relax and so accommodate itself to elevated tension. This phenomenon of relaxation undoubtedly also occurs to some degree in closed intestinal loops, but it is evident that sooner or later the elevated pressure must become constant, bringing with it the danger of strangulation.

EFFECT OF DISTENTION ON INTESTINAL SECRETION

Enderlen and Hotz[19] in 1911 noted that in the later stages of obstruction there was extravasation of fluid into the bowel. More

19. Enderlen, E., and Hotz, G.: Ueber die Resorption bei Ileus und Peritonitis, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **23**:755, 1911.

recently Herrin and Meek,[20] working with various types of intestinal fistulas, obtained a tremendous increase in secretion by distending the pouches with balloons at a tension of 80 mm. of mercury. They obtained as much as 550 cc. in twenty-four hours from a pouch 20 cm. in length. They stated: "Distention is a strong stimulus to intestinal secretion, and in obstruction this must work in a vicious cycle." This increase in secretion may be brought about in two ways:

1. By some nervous reflex mechanism whereby, as a result of the increased activity incident to distention, glandular secretion is increased by stimulation through Auerbach's plexus.

2. By increase in capillary permeability. It has been shown by Landis[21] that increase of capillary pressure results in increased permeability of the capillary wall. Experimentally, stasis of blood also increases the permeability of the capillary wall, so that at first a protein-free filtrate and later a protein-containing filtrate passes out of the blood vessels into the tissue spaces. Both increased intracapillary pressure and stasis of blood occur in simple intestinal obstruction. From the capillaries in the mucosa fluid passes into the lumen of the bowel, and from the serosa fluid passes into the peritoneal cavity. With active peristalsis sufficiently strong to result in local anemia of the intestine, fluid may actually be squeezed out of the capillaries.

Burget[4] in 1930 noted that the severity of symptoms in dogs with closed loop obstructions varied directly with the degree of distention of the loops. If the distention was allowed to increase, anorexia, vomiting and dehydration appeared. Relief of distention by aspiration of the contents of the loops at once relieved the symptoms. Herrin and Meek[20] have recently demonstrated that a nervous cause is largely responsible for this train of events. Dogs were able to withstand distention of denervated loops of bowel indefinitely. They did not lose their appetites and were able to maintain the level of their blood chlorides in spite of an abnormal loss of fluid and electrolytes from the distended ileal loops. Dogs in which the nerve supply of the bowel was not interfered with died showing the clinical picture of high intestinal obstruction—that is, dechlorination of the tissues and of the blood, and dehydration.

The degree to which this increased amount of fluid in the bowel contributes to distention is dependent on the level of obstruction. With high obstruction, when the fluid is evacuated early by vomiting, the distention is much less than when the obstruction is in the lower part

20. Herrin, R. C., and Meek, W. J.: Distention as a Factor in Intestinal Obstruction, Arch. Int. Med. **51**:152-168 (Jan.) 1933.

21. Landis, E. M.: Micro-Injection Studies of Capillary Permeability: The Effect of Lack of Oxygen on the Permeability of the Capillary Wall to Fluid and to Plasma Protein. Am. J. Physiol. **83**:528-542. 1928.

of the ileum. In low ileal obstruction, especially in obstruction of the closed loop type which occurs in the colon because of the competent ileocecal sphincter, the bowel cannot decompress itself by reversed peristalsis and regurgitation; thus, the increased secretion results in progressive augmentation of the distention.

Under the influence of obstruction, the total amount of fluid in the lumen of the bowel is increased by the response of the intestine to distention. Since the intestine fails to reabsorb this fluid, the cause of dehydration is apparent.

INFLUENCE OF OBSTRUCTION AND INCREASED INTRAENTERIC PRESSURE ON INTESTINAL ABSORPTION

It is generally conceded that the contents of both the obstructed and the normal bowel are toxic if injected intravenously. The manner and the routes of toxic absorption from the obstructed bowel are still subjects of considerable speculation and debate. The possible avenues by which toxic material of any kind may be absorbed are (1) the mesenteric veins, (2) the lymphatics and (3) the intestinal wall (transperitoneally by diffusion).

Venous Absorption.—Decreased absorption through the mesenteric veins of such substances as dextrose, salt, iodide, strychnine and histamine has been observed by many investigators. It is remotely possible, however, that the factor of selective absorption may play a role in determining the degree of absorption through the veins in simple intestinal obstruction. It may be that the bowel in a condition of obstruction becomes permeable to a toxin which is different from any of the substances used in experiments on animals.

This decrease of absorption, both quantitative and qualitative, has been attributed to the increase of intraenteric pressure which occurs in intestinal obstruction. Gatch and Culbertson [22] have investigated the absorption of specific substances, such as bromides and alcohol, from the normal bowel under moderately increased intraenteric pressure. They found that for pressures between 10 mm. of mercury and the diastolic blood pressure of the animals used in the experiments the rate of absorption of these substances is relatively constant.

The deleterious effect of simple ileal obstruction on the absorption of water from loops of ileum is illustrated by the fact that under identical experimental conditions closed loops of normal ileum absorbed approximately 90 per cent of the amount of water necessary to fill them, while loops of ileum which had been subjected to previous obstruction were able to absorb less than 10 per cent (table 11).

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22. Gatch, W. D., and Culbertson, C. G.: Circulatory Disturbances Caused by Intestinal Obstruction, Ann. Surg. **102**:619-635, 1935.

Absorption of Water from the Normal and from the Obstructed Ileum at Various Levels of Increased Pressure: There is little information available on the effects of relatively low intraenteric pressure, such as exists in experimental and clinical intestinal obstruction on absorption either from the normal or from the obstructed bowel. To supply these data the following experiments were undertaken:

In 41 normal dogs the amount of water absorbed from equal-sized loops of the lower portion of the ileum under the influence of a pressure of 10 to 100 cm. of water for one hour was determined. In this series of experiments on normal dogs it was found that a pressure of 40 cm. of water was the optimal pressure for absorption and that increase of pressure above that level resulted in lowering of the absorption ratio (table 12). A pressure of 40 cm. of water is equivalent to a pressure of about 3 cm. of mercury, which pressure Van Zwalenberg [23] has shown to be the level at which the circulation of the intestinal wall begins to be impaired.

TABLE 11.—*Absorption of Water from the Normal and from the Obstructed Ileum*

Dog	Weight, Lb.	Amount Injected, Cc.	Amount Absorbed, Cc.	Percentage Absorbed
1 (normal)	44	50	47	94.0
2 (normal)	41	30	26	86.6
3 (normal)	28	50	42	84.0
4 (normal)	54	40	33	82.5
Average normal				86.7
5 (with obstruction)	19	60	5	8.3
6 (with obstruction)	14	60	6	10.0
Average with obstruction				9.0

The time factor period of observation (one hour) in these experiments is most important, because when a pressure of 40 cm. of water was sustained over a considerably longer period (seventeen hours) definite pathologic changes in the wall of the bowel occurred. No such changes could be demonstrated in the intestinal wall after distention by a pressure of 40 cm. of water for one hour.

In a series of experiments on dogs with simple ileal obstruction of forty-eight hours' duration, a similar result was obtained; 40 cm. of water was the optimal pressure for absorption, but with higher pressures the decrease in absorption was more marked than in the series of experiments on normal dogs (table 13).

There was a significant decrease in absorption of water from the obstructed ileum as compared with its absorption from the normal ileum, varying between 20 to 60 cm. of water (table 14 and fig. 5). There was no significant difference between the two groups in the amount of water absorbed at a pressure of 10 cm. Pressures above 80 cm. of water were not utilized in this series because at these higher levels of pressure tearing of the serosa and leakage of the intestinal contents occurred.

23. Van Zwalenberg, C.: Strangulation Resulting from Distention of the Hollow Viscera: Its Bearing upon Appendicitis, Strangulated Hernia and Gall Bladder Disease, Ann. Surg. **46**:780-786, 1907..

This amount of pressure (40 cm.) is of course the optimal level for the absorption of water only for the particular segment of the bowel investigated (the lower part of the ileum). From the work of

TABLE 12.—*Effect of Increased Intraenteric Pressure on Absorption of Water from Loops of Normal Ileum*

	Dog	Weight, Kg.	Amount Absorbed, Cc.	Ratio, Cc. per Kg. per Hour
10 cm. water	1	15.0	100	6.6
	2	22.7	105	4.6
	3	26.4	165	6.2
	4	26.4	125	4.7
	5	12.3	70	5.7
	6	15.0	95	6.3
	7	19.5	115	5.9
	8	16.8	70	4.2
	Average ratio at 10 cm.			5.5
20 cm. water	9	20.4	236	11.5
	10	20.9	190	9.1
	11	14.1	180	12.7
	12	13.2	120	9.1
	13	15.4	130	8.8
	14	9.1	125	13.7
	15	7.3	60	8.2
	16	12.7	160	12.6
	17	11.4	100	8.8
	Average ratio at 20 cm.			10.5
40 cm. water	18	19.1	285	14.9
	19	14.1	170	12.1
	20	10.0	150	15.0
	21	12.4	130	10.5
	22	17.7	190	10.7
	23	9.1	110	12.1
	24	8.6	115	13.4
	Average ratio at 40 cm.			12.7
60 cm. water	25	20.5	210	10.2
	26	17.3	125	7.2
	27	12.7	160	12.6
	28	20.5	150	7.3
	29	10.0	95	9.5
	30	12.3	120	9.7
	Average ratio at 60 cm.			9.4
80 cm. water	31	6.8	55	8.1
	32	9.5	50	5.3
	33	9.5	70	7.4
	34	15.0	50	3.3
	35	18.2	120	6.6
	Average ratio at 80 cm.			6.1
100 cm. water	36	11.4	55	4.8
	37	12.3	35	2.8
	38	14.5	72	4.9
	39	17.3	105	6.0
	40	11.8	75	6.3
	Average ratio at 100 cm.			4.9

Dragstedt,[24] it may be surmised that the optimal level is lower in the jejunum and higher in the proximal portion of the colon, as he has

24. Dragstedt, C. A.; Lang, V. F., and Millet, R. F.: Relative Effects of Distention on Different Portions of the Intestine, Arch. Surg. **18**:2257-2263 (June) 1929.

shown that from the jejunum to the colon a correspondingly higher intraenteric pressure is necessary to interfere with circulation to the wall of the bowel.

TABLE 13.—*Effect of Increased Intraenteric Pressure on Absorption of Water from Loops of Obstructed Ileum*

	Dog	Weight, Kg.	Amount Absorbed, Cc.	Ratio, Cc. per Kg. per Hour
10 cm. water	1	13.4	100	7.5
	2	11.6	100	8.6
	3	19.5	110	5.6
	4	16.9	80	4.7
	5	16.4	75	4.6
	Average ratio at 10 cm.			6.2
20 cm. water	6	32.5	170	5.3
	7	20.0	150	7.5
	8	19.5	200	10.2
	9	19.5	80	4.1
	10	9.1	55	6.0
	Average ratio at 20 cm.			6.6
40 cm. water	11	15.0	160	10.6
	12	20.0	190	9.5
	13	14.1	160	11.3
	14	15.0	150	10.0
	Average ratio at 40 cm.			10.4
60 cm. water	15	13.2	82	6.2
	16	17.3	55	3.2
	17	28.6	155	5.3
	18	21.8	85	3.9
	19	23.2	85	3.7
	Average ratio at 60 cm.			4.4
80 cm. water	20	12.0	50	4.1
	21	20.5	55	2.7
	22	21.0	30	1.4
	23	21.0	30	1.4
	24	10.1	35	3.3
	25	14.0	20	1.5
	Average ratio at 80 cm.			2.4

TABLE 14.—*Comparison of Effect of Obstruction of the Ileum on the Absorption Ratio, by Fisher's Method*

Pressure	Ratio		Differences	t	P
	Normal	Obstructed			
10 cm.	5.53	6.20	+0.67	0.92	0.40
20 cm.	10.5	6.62	−3.88	3.18	<0.01
40 cm.	12.67	10.35	−2.32	2.37	0.05
60 cm.	9.42	4.46	−4.96	4.77	<0.01
80 cm.	6.14	2.4	−3.74	1.28	0.20

These findings are in agreement with those of early workers, Hamburger [25] and Leubuscher,[26] on the effect of intraintestinal pressure

25. Hamburger, H. J., cited by Goldschmidt, S.: On the Mechanism of Absorption from the Intestine, Physiol. Rev. **1**:421-453, 1921.

26. Leubuscher, cited by Goldschmidt, S.: On the Mechanism of Absorption from the Intestine, Physiol. Rev. **1**:421-453, 1921.

on intestinal absorption. Leubuscher ascribed the primary increase of absorption to unfolding of the mucosa and the secondary decrease to interference with the blood supply. Wells [27] found that the absorption of saline solution from closed loops was in direct proportion to the intraenteric pressure. However, his studies include pressures only up to 16 cm. of water. Elman and Aird,[6] in two experiments on animals and in one observation on a human being, showed essentially the same result which was obtained in these experiments. They noted maximum absorption at 50 cm. of water. At higher pressures (85 to 90 cm. of water), absorption did not take place. In their single experiment on a human being, the maximum absorption occurred at a pressure of

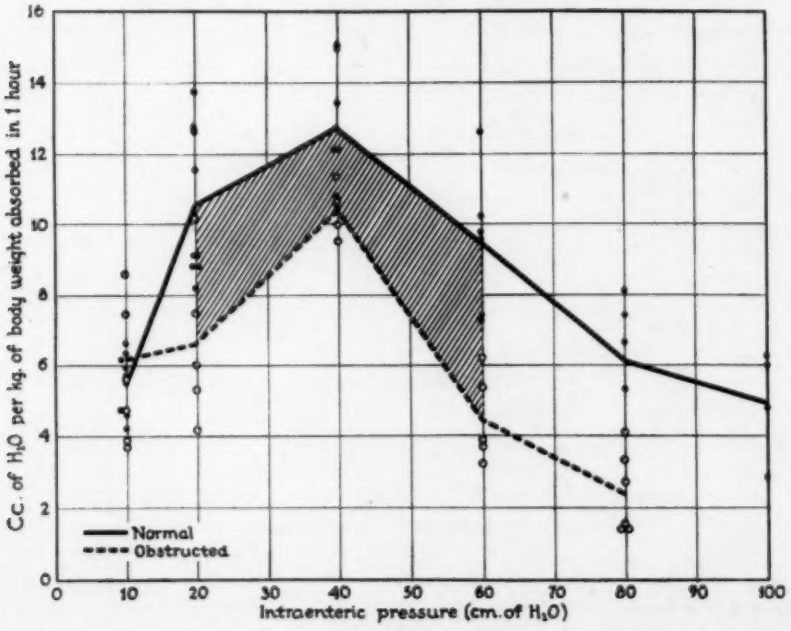

Fig. 5.—Effect of increased intraenteric pressure on the absorption of water from the normal and from the obstructed ileum. The shaded areas indicate significant difference between means.

40 cm. of water and fell progressively on increase of pressure until at a pressure of 70 cm. of water definite secretion into the loop occurred.

Absorption of Strychnine from the Normal and From the Obstructed Ileum: Though the avenues by which absorption may occur in obstruction are known, it is extremely difficult to ascertain definitely through

27. Wells, H. S.: Passage of Materials Through Intestinal Wall: Relation Between Intra-Intestinal Pressure and the Rate of Absorption of Water, Am. J. Physiol. **99**:209-220, 1931.

which avenue and to what degree toxic absorption, if any, takes place. It is possible by the employment of such substances as strychnine and organic dyes and by the elimination of certain of the avenues for absorption to gain some information on this subject.

Strychnine was absorbed directly from the peritoneal cavity of the cat in two minutes and from the loops of the normal ileum of the cat in three minutes. In animals with intestinal obstruction a slight but definite delay in absorption from the bowel was evident within the first twenty-four hours of obstruction; that is, three to six minutes were required for absorption as compared with two to three minutes in the normal intestine. This increase of absorption time was more marked when the loops were outside the peritoneal cavity (eight to thirteen minutes). The absorption time seemed to increase directly with the duration of the obstruction.

Absorption of strychnine takes place very rapidly through the mesentery even when the loop is outside the peritoneal cavity. The comparatively slight increase of absorption time in obstructed loops in the extraperitoneal position is due to the abnormal location of the loop, to slight tension on the mesentery, to handling of the bowel or to changes of temperature which must occur despite attempts to maintain normal conditions. It appears that the obstructed bowel is more sensitive to such changes than is the normal bowel.

Influence of Increased Intraenteric Pressure on the Absorption of Strychnine from the Normal and from the Obstructed Ileum: To determine the level of intraenteric pressure which prevents intestinal absorption, experiments similar to those of Gatch [28] were carried out. With a constant pressure of more than 100 mm. of mercury, absorption of strychnine could not be demonstrated during several hours of observation. Only when the intraenteric pressure was reduced to approximately 70 mm. of mercury could the absorption of strychnine be shown. This occurred regardless of the peritoneal or extraperitoneal location of the loop, indicating that absorption took place by way of the mesentery. Two dogs with simple low ileal obstruction of three days' duration showed symptoms of absorption of strychnine at pressures of 50 and 60 mm. of mercury respectively when this experiment was carried out.

When strychnine was introduced into a loop a pressure of 100 mm. of mercury and the abdomen was closed, convulsions did not develop until the intraenteric pressure was decreased by relaxation of the bowel to a pressure of 75 mm. of mercury, at which critical point the strychnine was absorbed via the veins of the mesentery.

It is concluded from these experiments that an intraenteric pressure of more than 75 mm. of mercury prevents the mesenteric absorption of strychnine.

It has been shown that absorption of strychnine via the mesenteric veins is less in simple ileal obstruction than under normal conditions.

28. Gatch, W. D.; Owen, J. E., and Trusler, H. M.: The Effect of Distention of the Bowel upon Its Circulation and upon Absorption from Its Lumen, West. J. Surg. **40**:161-167, 1932.

The obstructed bowel absorbs only a fraction of the amount of water taken up by a similar segment of normal intestine under identical experimental conditions. Increase of the intraintestinal pressure results in a progressive increase in the absorption of water from the normal bowel until a pressure of 40 cm. of water is reached. Higher pressures produce a fall of the rate of absorption to below the normal rate. The same holds true, but to a lesser degree, in the obstructed bowel. Pressures above 75 mm. of mercury effectively prevent venous absorption.

Lymphatic Absorption.—An attempt was made to determine the occurrence of lymphatic absorption in simple intestinal obstruction and with increased intraenteric pressures.[29]

Absorption of Dyes Via the Lymphatics in Simple Ileal Obstruction: Fifteen normal cats were given trypan blue by stomach tube and 5 by enteral injections. In none was the dye visible in the regional lymph nodes at intervals of two to forty-eight hours. When cats with simple ileal obstruction were given the dye by stomach tube none appeared in the lymphatics. When this dye was injected into the bowel at the time the obstruction was produced, 3 of 8 cats showed colored nodes. When intestinal stasis was induced by chemical peritonitis (with iodine) 1 of 5 cats showed blue lymph nodes.

Absorption of Dyes from the Lumen of the Intestine Under Increased Intraenteric Pressure: Closed ileal loops were prepared in 12 cats and the loops distended with dye until the walls became tense (marked increase of intraenteric pressure). Ten of the 12 cats at autopsy (twenty-four to seventy-two hours) showed blue nodes. In several of these experiments the dye, as it was injected into the lumen of the bowel under pressure, could be seen to enter Peyer's patches after five or ten minutes and subsequently to enter the lymph vessels of the mesentery. This procedure was repeated in vitro with fresh excised viable segments of normal and obstructed bowel of the cat, with the following results: Pressures of 100 mm. of mercury in a normal loop forced the dye out into the lymphatics in five minutes. In another normal loop a pressure of 50 mm. of mercury forced the dye into the lymph vessels in one hour. When a fresh viable loop excised from a cat with simple ileal obstruction of forty-eight hours' duration was subjected to a pressure of 40 mm. of mercury, dye appeared in the lymph vessels in thirty minutes. When nonviable loops were subjected to increased intraenteric pressure, the dye, in addition to being forced rapidly into the lymphatics, permeated the wall of the intestine directly (table 15).

Absorption of Bacteria Via the Lymphatics: The absorption of bacteria through the lymphatics was studied in the following manner: A heavy suspension of Bacillus pyocyaneus was injected into the ileums of 6 normal cats. Preliminary cultures of regional lymph nodes were sterile. Cultures of material taken from the nodes one hour after the intraenteric injection showed B. pyocyaneus in 2 cases and failed to do so in 4; in 2 of the latter, cultures of B. pyocyaneus were obtained when the cats were again operated on after twenty-four hours. In 2 additional cats in which the bacterial suspension was introduced into closed loops

29. Sperling, L., and Wangensteen, O. H.: Lymphatic Absorption in Simple Obstruction: Significance of Distention upon Its Occurrence, Proc. Soc. Exper. Biol. & Med. **33**:22-26, 1935.

under pressure for one hour, cultures of material taken from the regional lymph nodes produced B. pyocyaneus.

Similar experiments were carried out in normal dogs and in dogs with intestinal obstruction. Cultures of B. pyocyaneus were obtained in material from the regional lymph nodes in 2 of 9 experiments on normal dogs and in all of 6 experiments performed on dogs with low ileal obstruction of twenty-four to ninety-six hours' duration. Control cultures of material taken from the lymph nodes before the injection of the bacterial suspension failed in every case to show B. pyocyaneus. Cultures of the lymph in the thoracic ducts of 5 normal dogs and of 6 dogs with intestinal obstruction proved entirely sterile. Cultures of blood from the femoral veins and from the mesenteric vein draining the obstructed bowel were all sterile.

In the experiments on the absorption of dyes, the lymph nodes of the animals with closed ileal loops were stained more deeply than those of the animals with simple ileal obstruction. The percentage of colored nodes was higher in the group with closed loops than in that with simple ileal obstruction. This is probably due to the fact that a higher intraenteric pressure is developed in the closed loop.

TABLE 15.—*Effects of Distention on Lymphatic Absorption and Transperitoneal Passage of Dyes in Excised Loops of Normal and Obstructed Ileum in Cats*

Time After Death, Hr.	Obstruction, Hr.	Pressure, Mm. Hg	Viability	Dye in Lymph Nodes After Min.	Pemeability of Bowel Wall After Min.
0	None	100	Viable	5	Negative, 60
0	None	50	Viable	60	Negative, 60
0	24	130	Viable	10	Negative, 60
0	48	40	Viable	30	Negative, 60
6	24	100	None	5	Positive, 15
8	48	100	None	10	Positive, 30

In the control experiments the absorption of dye into the lymphatics is without doubt prevented by its rapid transport through the bowel. Transport, of course, does not take place in circumstances of obstruction. In its stead there are stasis, distention and increased intraenteric pressure.

The results of these experiments are in accord with the well known physiologic principle that increase of venous pressure enhances lymphatic flow. In the closed loop type of obstruction the increased tension in the bowel produces increased venous pressure. The appearance of dyes in the lymphatics of cats dying of simple intestinal obstruction and the failure to demonstrate the dye in the lymphatics of animals with simple intestinal obstruction that were killed may be explained by the fact, as was demonstrated by Bainbridge,[30] that the flow of lymph continues after death. From these experiments it is apparent that a

30. Bainbridge, F. A.: Postmortem Flow of Lymph, J. Physiol. **34**:275-281. 1906.

high correlation exists between the increased intraenteric pressure in obstruction and the degree of lymphatic absorption.

It appears that the regional lymph nodes normally serve as an effective barrier against the overwhelming absorption of bacteria into the blood stream via the mesenteric lymphatic channels. True bacteremia probably does not occur and is probably not the cause of death in uncomplicated intestinal obstruction, as cultures producing the test organism could not be obtained in material taken from the thoracic duct. The lymph nodes yielding the organisms acted as efficient barriers and prevented centripetal invasion. Toxemia of bacterial origin or from lymphatic absorption of toxic amines from the bowel, however, has not been wholly excluded.

Significance of Lymphatic Absorption in Simple Intestinal Obstruction: An attempt was made to evaluate the significance of lymphatic absorption in the lethal issue of simple intestinal obstruction by interruption of the lymphatic channels. This was accomplished by ligating and cutting the lymphatic pedicle of the mesenteries in 4 cats after the establishment of simple ileal obstruction. The period of survival was not greater than that of the control animals, in which the lymphatic pedicle was not severed.

These observations seem to justify the conclusion that intestinal obstruction and increased intraenteric pressure increased the absorption of dyes and of bacteria into the regional lymph nodes. No evidence was, however, adduced to indicate that this occurrence is of great significance in the lethal issue of simple intestinal obstruction in animals.

Transperitoneal Absorption.—The wall of the normal viable bowel is resistant to the passage of toxic material from its lumen into the peritoneal cavity. Most investigators are agreed that transperitoneal passage of any material does not take place unless extreme increases of intraintestinal pressure obtain or unless gross injury to the bowel wall is present. Schönbauer,[31] Gatch,[28] Chenuth,[32] and others have advanced the hypothesis that death following intestinal obstruction is due to the absorption of toxins which have passed through the wall of the bowel into the peritoneal cavity.

Stone and Firor,[5] Gatch,[28] Dobyns and Dragstedt[33] and others have indicated that increased intraenteric pressure may force toxic materials through the intestinal wall. Gatch's work on the permeability of the bowel to hydrocyanic acid under increased tension was repeated

31. Schönbauer, L.: Die Fermente in ihrer Beziehung zu gewissen Erkrankungen der Gallenblase und zum Ileus, Arch. f. klin. Chir. **130**:427-462, 1924.

32. Chenuth, A.: L'experimentation dans l'occlusion mechanique du jejuno-ileum, Rev. de chir., Paris **64**:474-834, 1926.

33. Dobyns, G. J., and Dragstedt, C. A.: Intra-Intestinal Pressure and Absorption from the Intestine, Proc. Soc. Exper. Biol. & Med. **30**:707-709, 1933.

and confirmed.[34] Apparently, however, the wall of the viable bowel is permeable by simple chemical compounds only, as Gatch was unable to show transperitoneal absorption of nicotine. The passage of strychnine through the wall of the viable bowel could not be demonstrated even with marked increase of intraintestinal pressure.

Passage of Strychnine Through the Wall of the Devascularized Bowel and Through Devascularized Bowel Which Had Previously Been Subjected to Simple Ileal Obstruction: In 4 cats with simple ileal obstruction of ninety-six hours' duration and in 1 cat with simple ileal obstruction of one hundred and forty-four hours' duration, the distal 3 inches (7.5 cm.) of the obstructed intestine was devascularized and a large dose of strychnine (10 to 50 mg.) was injected into the lumen of this segment. Similar devascularized loops were prepared in 5 normal cats. After closure of the abdomen, the cats were revived and were observed for signs of absorption of the poison. The animals with ileal obstruction showed convulsive signs within two and one-half or three hours and were dead within three or four hours. The normal cats did not show signs of poisoning until from four and one-half to seven hours had elapsed.

Effect of Increased Intraintestinal Pressure on the Passage of Strychnine Through the Wall of the Devascularized Bowel and Through the Bowel Which Had Previously Been Subjected to Simple Ileal Obstruction: Closed loops of ileum 10 inches (25 cm.) in length were prepared in 4 normal dogs. The loops were devascularized, and 50 mg. of strychnine was introduced into the lumens. Two of the loops were distended with air to a pressure above 100 mm. of mercury; 2 others were not distended. The dogs were revived and were observed carefully for signs of strychnine poisoning. The dogs with ileal loops under increased pressure had convulsions within three hours. Those in which the ileal loops were not under pressure remained free from symptoms for more than eight hours.

In 3 dogs with simple ileal obstruction of five days' duration closed loops of ileum were prepared just above the site of obstruction. The intraenteric pressure was raised to approximately 120 mm. of mercury, and 50 mg. of strychnine was introduced into the lumen of the closed loop. One dog showed signs of strychnine poisoning within eighteen minutes. The second dog had symptoms within forty-five minutes. The third dog showed no symptoms after seventy-five minutes. Examination revealed numerous rents in the serosa in all 3 cases. The obstructed bowel cannot tolerate great increases of intraenteric pressure for any considerable time. In all of the experiments in which obstructed bowel was utilized and subjected to a pressure of 80 mm. of mercury or more for one hour, there developed rents on the antimesenteric border which might facilitate transperitoneal passage of poisons or toxins.

Effect of Increased Intraenteric Pressure on the Passage of Dyes Through the Wall of the Bowel: Freshly excised viable loops of ileum were obtained immediately after death from 2 normal cats killed during other experiments.[34] The loops were filled with a 1 per cent solution of trypan blue and subjected to 50 and 100 mm. of mercury of pressure, respectively, for one hour. Freshly excised loops from 2 cats with an antecedent low ileal obstruction of twenty-four and forty-eight hours' duration, respectively, were filled with dye and subjected to distention

34. Sperling, L., and Wangensteen, O. H.: Transperitoneal Absorption: VI. The Significance of Impaired Viability and the Influence of Distention on Its Occurrence, Proc. Soc. Exper. Biol. & Med. **32**:1385-1389, 1935.

at respective pressures of 40 and 130 mm. of mercury. The dye had not permeated the bowel after one hour of distention. However, when ileal loops from 2 cats with intestinal obstruction, dead six and eight hours respectively, were subjected to distention at a pressure of 100 mm. of mercury, the nonviable bowel permitted the dye to pass directly through its wall in fifteen to thirty minutes. When a 1 per cent solution of gentian violet was injected into the intestinal lumen in cats with simple obstruction and obstruction of the closed loop type, only a slight staining of the mucosa was present in a few instances on microscopic section of the bowel (Churchman's method[35]). As this was not a constant finding and was present only in cats dead several hours, it was assumed that the penetration of the mucosa was a postmortem phenomenon. In none of the cats could transperitoneal passage of the dye be demonstrated by wrapping gauze about the outside of the bowel. In 1 experiment the dye was introduced into the loop under high pressure so that the loop was very tense. When the cat was killed seventy-two hours later (moribund), several purple spots were visible on the antimesenteric border. These were judged to be nonviable necrotic areas through which the dye had passed.

Correlation of the Viability of the Intestinal Wall with Its Permeability in Simple Ileal Obstruction: The following experiments were undertaken to determine whether any correlation exists between the permeability and the viability of the bowel in simple ileal obstruction.

Fifteen cats with simple ileal obstruction of six to one hundred and sixty-eight hours' duration were killed at definite intervals after the obstruction, and the viability and permeability of the loops were noted in the following manner: The terminal 4 inches (10 cm.) of the obstructed bowel was removed, and the closed segment was at once immersed in a beaker of Ringer's solution (temperature 37 C.). The viability of the segment was determined by noting the length of time during which it reacted by contraction to a measured electrical stimulus repeated every fifteen minutes. The loop was considered to have lost its viability when no contraction appeared after prolonged stimulation. Permeability of the bowel was determined by immersing the segments, filled with potassium ferrocyanide, at intervals of fifteen minutes into another beaker of Ringer's solution, containing ferric chloride, and noting the time at which the characteristic prussian blue color appeared.

The segments of bowel from the cats with intestinal obstruction were all viable and showed no permeability when first removed. They remained viable for periods varying between fifteen minutes to one and one-quarter hours after the animals were killed. Most specimens resisted permeation for some time after some impairment of viability was evident. The segments from the dead cats which had been normal and from cats already dead of intestinal obstruction were of course nonviable and showed immediate permeation. Loops obstructed for ninety-six hours or longer showed signs of impairment of viability (table 16).

In a similar study, Schempp[36] could not find any definite relation between the duration of obstruction and the viability of the bowel.

That a real difference in permeability and viability exists between normal bowel and obstructed bowel is shown by experiments on the

35. Churchman, J. W.: The Selective Bacteriostatic Action of Gentian Violet and Other Dyes, J. Urol. **11**:1-18, 1924.

36. Schempp, E.: Zur Frage der Durchlässigkeit der Darmwand für gelöste Stoff, insbesondere beim Darmverschluss, Beitr. z. klin. Chir. **143**:728-736, 1928.

permeability of devascularized loops by strychnine. Obstructed devascularized loops become permeable in two and one-half or three hours, while the normal loops resist permeation at least four hours. Under similar conditions of increased pressure, the bowel of dogs with intestinal obstruction becomes permeable somewhat earlier because of tearing of the serosa.

TABLE 16.—*Correlation of Viability of Intestinal Wall in Simple Ileal Obstruction with Its Permeability by Potassium Ferrocyanide*

Duration of Obstruction, Hr.	Viability Impaired After Min.	Viability Lost After Min.	Permeable After Min.
8 normal cats	30-75	30-150	15-55
6, 18, 18, 24	30-60	60-120	45-60
30, 36, 48, 48	30-50	45-75	15-60
72, 72	15-30	30-90	30-90
96	15	15	25
120	15	30	20
168	15	30	5
Normal loop for cat dead 8 hours			5
Loop 72 hours obstructed from cat dead 8 hours			6
Loop 96 hours obstructed from cat dead 8 hours			2

TABLE 17.—*Effect of Sustained Intraenteric Pressure on the Appearance, Permeability and Viability of the Ileum*

Dog	Pressure Applied, Cm.	Reoperated On, After Hr.	Results
1	10	28	Petechial hemorrhage; viable; not permeable
2	10	27	Petechial hemorrhage; hemorrhage into lumen; viable; not permeable
3	15	18	Congested; hemorrhage into lumen; early necrosis; viable; not permeable
4	20	10	Congested; viable; not permeable
		22	Congestion marked; impaired viability; not permeable
		28	Hemorrhagic peritoneal fluid; necrosis; nonviable; permeable
5	20	22	Congested; viable; not permeable
		32	Dog dead; gangrene and necrosis; nonviable; permeable
6	40	2	Negative; viable; not permeable
7	40	4	Hemorrhage; viable; not permeable
8	40	11	Congested; bloody peritoneal fluid; viable; not permeable
		17	Dog dead; areas of necrosis; nonviable; permeable
9	40	28	Necrosis; nonviable; permeable
10	40	20	Split serosa; gangrenous patch; nonviable; permeable
11	40	20	Necrosis; nonviable; permeable
12	40	20	Hemorrhagic areas; early necrosis; viable; not permeable
		24	Necrosis spreading; nonviable; not permeable
		30	Mucosal ulcers; nonviable; permeable

Results of experiments on diffusion of dyes through the wall of the bowel accord with those of experiments on absorption of strychnine, that is, viable bowel resists permeation and nonviable, or dead, bowel is readily permeable.

The work of Gatch,[22] Dragstedt [33] and others concerning the effect of increased intraintestinal pressure on absorption is substantiated. The greater potential for the occurrence of transperitoneal absorption in the obstructed bowel is well shown in the results accompanying devasculari-

zation of normal and previously obstructed bowel. Still, evidence of transperitoneal absorption does not ordinarily appear until there is anatomic evidence of damage to the wall of the intestine, as will be shown in some following experiments.

These observations appear to warrant the conclusion that transperitoneal absorption in simple intestinal obstruction may occur, but only through devitalized segments of bowel subjected to distention, and that the obstructed bowel under similar experimental conditions is more permeable to strychnine than is the normal bowel.

EFFECT OF PROLONGED DISTENTION ON THE STRUCTURE, VIABILITY AND PERMEABILITY OF THE WALL OF THE BOWEL

Any pathologic changes in the bowel as a consequence of increased intraenteric pressure must be secondary to the effect of this increased pressure on the circulation of the intestinal wall.

Kocher [37] was the first to recognize the significance of increased intraenteric pressure. Many American investigators have since referred to the obvious disturbance of circulation and consequent impairment of function of the intestine as a result of increased intraenteric pressure (Van Zwalenberg,[38] Dragstedt,[33] Gatch and others).

Van Zwalenberg [39] attempted to explain the pathologic changes occurring in intestinal obstruction on the basis of anoxemia of the wall of the bowel. According to his research, stasis of blood results in a decreased oxygen supply, which affords excellent opportunity for the development of infection and its sequelae, gangrene, necrosis and perforation of the intestinal wall. He cited experimental evidence presented by Burget and Visscher [40] to show that the bowel deprived of its blood supply uses up all available oxygen very rapidly. Bacteria, especially anaerobes and facultative anaerobes, grow well in asphyxiated tissue. There can be no doubt that partial asphyxia of the wall of the bowel, due to stasis of blood, occurs in simple intestinal obstruction.

The stagnant contents of obstructed loops form an excellent culture medium for the growth of bacteria. Thus, bacterial growth may invade the partially asphyxiated tissues of the bowel, with resulting pathologic

37. Kocher, T.: Ueber Ileus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **4**:195-230, 1899.

38. Van Zwalenberg, C.: Hydraulic Vicious Circle as It Develops in Intestine: Effect of Intra-Intestinal Pressure on the Pathology and Physiology of the Bowel, Am. J. Surg. **18**:104-112, 1932.

39. Van Zwalenberg, C.: Hydraulic Vicious Circle as It Develops in Acute Appendicitis, Am. J. Surg. **16**:427-440, 1932.

40. Burget and Visscher, cited by Van Zwalenberg, C.: Oxygen Lack in Hydraulic Vicious Circle, Am. J. Surg. **18**:133, 1932.

changes. Downey [41] in 1917 demonstrated that venous stasis is conducive to invasion by leukocytes. This would explain the infiltration of the bowel with polymorphonuclear and mononuclear cells which occurs when the intestine is subjected to prolonged distention. Thus, infiltration with leukocytes occurs first as a response to the invasion of the partially asphyxiated tissues by bacteria and second as a result of the stasis of blood, which in itself is conducive to chemotaxis of leukocytes.

Experiments were carried out to ascertain what grade of sustained intraenteric tension is necessary to produce anatomic changes in the wall of the bowel. The effects of such distention on its viability and its permeability were simultaneously studied.

Higher levels of increased intraenteric pressure resulted in correspondingly greater degrees of pathologic phenomena. Closed ileal loops subjected to a constant

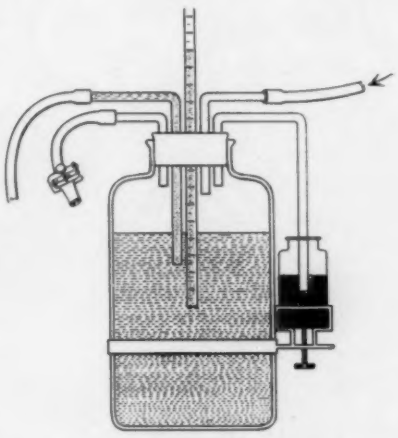

Fig. 6.—Constant pressure bottle of Perusse.

pressure [42] (fig. 6) of 10 cm. of water for twenty-eight hours revealed congestion of the bowel with petechial hemorrhages on the antimesenteric border. There was no marked change in the diameter of the bowel. Viability was not impaired, since active contraction followed stimulation with the faradic current. Permeation of the wall of the bowel by potassium ferrocyanide could not be demonstrated. A loop subjected to a pressure of 15 cm. of water for eighteen hours was much congested; there were hemorrhage into the lumen of the bowel and gangrenous patches on its antimesenteric border. Loops subjected to a pressure of 20 cm. of water for a similar period were dilated to two or three times the normal size and showed congestion of the wall of the entire bowel, with hemorrhagic infarcts on the antimesenteric border, and areas of early necrosis through which penetration of potassium ferrocyanide could be demonstrated. A pressure of

41. Downey, H.: Reaction of Blood and Tissue Cells to Acid Colloidal Dyes Under Experimental Conditions, Anat. Rec. **12**:429-448, 1917.

42. Perusse, G. L. Jr.: The Solution of Choice in Proctoclysis, Surg., Gynec. & Obst. **54**:770-784, 1932.

40 cm. of water was tolerated for only seventeen hours before gangrene and necrosis ensued. At this pressure the loops were dilated and invariably showed large gangrenous areas with loss of viability and abnormal permeability. Loops of colon subjected to pressures of 30 and 50 cm. of water for twenty-four hours showed similar changes. Loss of viability was encountered only in necrotic areas. Abnormal permeability of the bowel was present through these areas, but never through viable bowel. It is significant that in 1 of these experiments the bowel did not become permeable until six hours after loss of viability was demonstrated (table 11).

Fig. 7.—Dog's ileum subjected to a pressure of 15 cm. of water for eighteen hours. Note hemorrhage into the lumen of the bowel, submucosal hemorrhage and infiltrated and necrotic areas in the muscular layers.

The microscopic observations (figs. 7, 8, 9 and 10) were as follows:

1. Congestion and dilatation of capillaries at a pressure of 10 cm. of water.

2. Extravasation of blood into the tissue spaces and into the lumen of the bowel at a pressure of 15 and 20 cm. of water.

3. Early mononuclear cellular infiltration in the submucosa, even at a pressure of 10 cm. of water.

4. Areas of gangrenous and necrotic tissue in all layers after twenty-four hours of distention at pressures of 20 or 40 cm. of water.

5. Infiltration of all layers of the bowel wall by polymorphonuclear leukocytes, especially concentrated in areas of necrosis.

Repeated tests of the animals' serums gave uniformly negative reactions to injection of prussian blue, indicating that potassium ferrocyanide is not absorbed from the lumen of the bowel even under conditions of increased tension. Positive results from a test of the serosal surface of the bowel indicated passage of the chemical directly through the wall of the bowel. When potassium ferrocyanide was injected

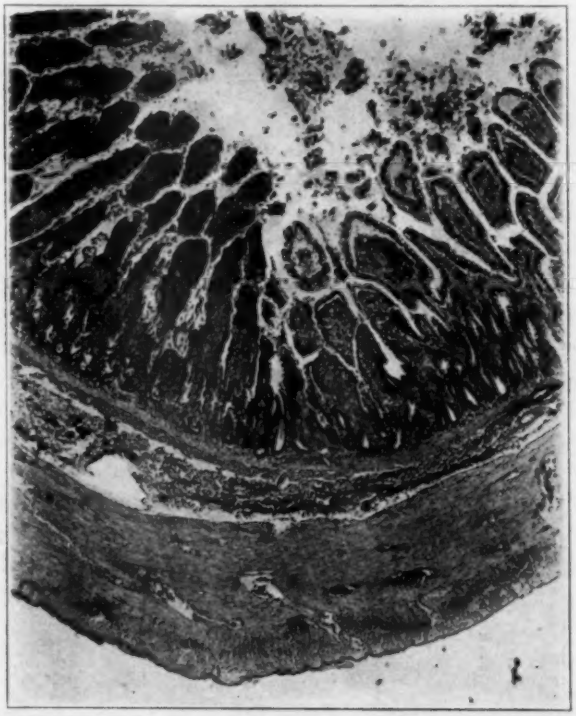

Fig. 8.—Dog's ileum subjected to a pressure of 20 cm. of water for thirty-two hours. Note hemorrhage into the lumen and necrotic areas in the submucosa and in the muscular layers.

intravenously in control experiments, the blood serum fifteen minutes later always gave a positive reaction.

The lesser anatomic effects observed at similar intervals in clinical cases as well as in experimental cases of simple obstruction of the small intestine, as contrasted with the results of a continuously maintained intraenteric pressure as related here, indicate that opportunity must exist in simple intestinal obstruction for occasional release from the pressures observed, which were regarded as sustained. Clinically, in obstruction of the colon (virtually a short closed loop, so made by the competent

Fig. 9.—Dog's ileum subjected to a pressure of 40 cm. of water for twenty hours. Note hemorrhage into the lumen and necrotic areas in the submucosa and the muscular layers.

Fig. 10.—Dog's colon subjected to a pressure of 50 cm. of water for twenty-four hours. Note areas of hemorrhagic infiltration and necrosis throughout all layers of the intestinal wall. (Section taken from the antimesenteric border.)

ileocecal sphincter) a closer counterpart of the effects of long-maintained intraenteric tension exists. The time necessary for the production of the anatomic and physiologic changes in such obstruction, moreover, is also considerably greater than that observed in these experiments—a fact which denotes that even in closed loop obstruction, in which the intraenteric pressure may be great, the intestine may, by dilating, protect itself to some extent from the effects of distention.

It is concluded from these experiments that:

1. There is a definite relation between the height of intraenteric pressure and the degree of pathologic change occurring after distention of the bowel.

2. Petechial hemorrhage can be demonstrated in the wall of the bowel when a constant pressure of 10 cm. of water is sustained over a sufficient period (twenty-four hours). A pressure of 20 cm. of water sustained for a similar period produces necrosis, loss of viability and abnormal permeability of the wall of the bowel. A sustained intraenteric pressure of 40 cm. of water will produce similar changes in seventeen hours.

3. Abnormal permeability of the intestinal wall does not occur unless its viability is impaired.

LETHAL FACTORS IN SIMPLE LOW ILEAL OBSTRUCTION

Any consideration of the factors contributing to the fatal outcome in low ileal obstruction must include all of the following:

1. Loss of fluid and of electrolytes by vomiting and by failure of the distended intestine to absorb a normal amount of fluid.

2. The effect of obstruction on the intestinal wall as it is related to (*a*) the mode of absorption of any toxic material from the lumen of the bowel, (*b*) alterations of the normal function of the bowel and, (*c*) impairment of the integrity of the intestinal wall.

No single factor is responsible for the fatal outcome in all cases, but death is probably due to any of several combinations of these factors.

The part played by dehydration in this type of obstruction varies considerably. When vomiting is profuse, dehydration and the associated alteration of the metabolites of the blood may be severe. However, these changes alone cannot account for the death of the animal, for, as previously indicated, the survival period of animals with low ileal obstruction usually cannot be materially lengthened by the administration of saline solution or Ringer's solution. The factor of loss of fluid assumes serious proportions only in cases of high obstruction of the small intestine. In these cases saline solution affords an effective substitute for the fluid lost.

Although many toxic substances have been isolated from the intestinal contents of animals with ileal obstruction, direct proof of their absorption and of the actual avenue of absorption is lacking. A tremendous amount of literature has accumulated dealing with the possible toxic factor, but as yet concrete evidence to the effect that a specific toxin is absorbed and is responsible for the fatal issue is not available. It is granted that the content of the obstructed bowel is toxic when injected intravenously, but so is the content of the normal bowel. The fact that such material, injected intravenously or by some other parenteral route, produces toxic symptoms does not prove anything more than that, for in carrying out such experiments the normal capacity of the mucosa to protect the organism against fatal infection and other menaces is entirely overlooked. If any toxin is absorbed through the mucosa, it may be by way of the lymphatics, since, as has been shown, absorption by all other normal avenues is decreased when the bowel is obstructed. It is significant that bacteria may be demonstrated in the mesenteric lymph nodes in far greater numbers in the obstructed bowel than in the normal bowel. Nevertheless, ligature of the lymphatic pedicle at the root of the mesentery does not prolong the survival of animals with simple intestinal obstruction.

Transperitoneal absorption must be considered a manifestation of impaired viability of the bowel which has been subjected to distention for a sufficient time to produce gross anatomic changes. Its occurrence is probably insignificant as long as the bowel remains viable, but it may become significant when a devitalized segment is subjected to prolonged distention. The intraenteric pressure, the duration of distention and the diameter of the intestinal coils determine the effect of the distention on the blood supply of the intestine and, in time, the presence or absence of gross anatomic changes and loss of viability of its wall which permit transperitoneal absorption to take place.

That the derangement of normal function which occurs at the level of obstruction is an important factor in the fatal issue is suggested by the following experiment.[43]

Ileosigmoidostomy was performed in 7 dogs in an attempt to alter the function of the terminal portion of the ileum by adapting it to the conditions of the colon as regards (1) stasis, (2) absorption of water and (3) bacterial flora. After periods varying between two and eight and one-half months, simple ileal obstruction was produced at the sites of the anastomoses. Three dogs survived for twenty-five, thirty-one and thirty-four days respectively. This period of survival is considerably longer than that of dogs with simple low ileal obstruction. A loss of 28 to 40 per cent of body weight occurred before death. Death was

43. Sperling, L., and Wangensteen, O. H.: Effect of Previous Ileosigmoidostomy on the Survival Period of Dogs with Low Ileal Obstruction, Proc. Soc. Exper. Biol. & Med. **31**:323-326, 1933.

apparently due to starvation, but it is significant that at autopsy structural changes, which in the light of previous experiments might conceivably permit transperitoneal absorption of a toxin, were evident in the wall of the bowel. These were areas of hemorrhagic necrosis on the antimesenteric border of the bowel, and in one case there was ulceration of the mucosa. Alteration of the chemical constituents of the blood was manifested by a reduced plasma chloride content and by elevation of the nonprotein nitrogen (table 18).

It is conceivable that in these experiments the ileum assumed the function of the colon, that is, the absorption of water, and accommodated itself to the conditions of the colon. Thus those functions which would ordinarily be lost to the dog in a case of low ileal obstruction because

TABLE 18.—*Summary: Ileosigmoidostomy Series*

Dog	Duration of Ileocolostomy, Mo.	Survival Period After Obstruction, Days	Saline Given	Weight Loss, %	Blood Chemistry Before Death*	Pathologic Picture
37	2	8	No	17.6	Cl. 412 U. 35.0	Distention and rubor of bowel wall
29	2	10	No	17.0	Cl. 305 U. 33.0 N.P.N. 65.0	Abscess about colon closure
45	3½	12	Yes	14.8	Cl. 264 U. 18.8 N.P.N. 37.2	Volvulus; segment of small bowel with strangulation
79	6	10	Yes	7.0	Cl. 379 U. 50.0 N.P.N. 80.0	Hemorrhagic necrosis on antimesenteric border
71	3½	25	Yes	28.0	Cl. 346 U. 11.5 N.P.N. 22.5	Hemorrhagic areas of necrosis on antimesenteric border
70	8	34	Yes	29.0	Cl. 346 N.P.N. 37.0	Closed loop formed by adhesions; hemorrhagic necrosis on antimesenteric border; ulceration of mucosa
74	8½	31	Yes	40.0	Cl. 478 N.P.N. 30.0	Hemorrhagic necrosis on antimesenteric border

* Cl. indicates chlorides; U., urea nitrogen; N.P.N., nonprotein nitrogen.

of the exclusion of the colon (below the obstruction) were preserved. The death of the dogs was then delayed until other factors, such as devitalization of the wall of the intestine due to progressive increase of distention, were sufficiently influential to produce abnormal permeability of its wall and to allow the transperitoneal absorption of some toxin.

COMMENT AND SUMMARY

Prior to 1930 a great deal of emphasis was placed on the alteration of the metabolites of the blood in intestinal obstruction, and there was a tendency to relegate the consideration of the local changes in the wall of the bowel to a position of secondary importance. That pathologic changes frequently occur in the wall of the bowel under conditions of obstruction and that they are important factors in the fatal issue has

since been stressed by many investigators. These changes are usually attributed to the impairment of the circulation of blood to the intestine by distention. Most of the recent experimental work has dealt with the effect of distention of the bowel on the intestinal circulation. It has been demonstrated that the flow of blood is decreased as the pressure within the intestine is increased (Dragstedt [24]). Many investigators have referred to the damaging effect of increased intraenteric pressure on the intestinal wall. There has been no previous work establishing the exact level of the intraenteric pressure necessary to produce such changes. In most of the experimental work reported heretofore, extremely high pressures, much higher than those which occur in clinical cases of intestinal obstruction, were utilized (Gatch [28]). From this study it appears that definite pathologic changes can be produced in the wall of the intestine by sustained pressures which are much lower than those used by other investigators and which are comparable to the levels actually measured in experimental and clinical cases of intestinal obstruction.

Observations were made of the effect on the wall of the bowel of sustained and increased intraenteric pressure of the degree measured in experimental and clinical cases of intestinal obstruction. It was determined that diffusion of potassium ferrocyanide, strychnine or dyes through the wall of the intestine into the peritoneal cavity does not occur as long as the intestine remains viable. Although a pressure of 10 cm. of water sustained for twenty-four hours produces petechial hemorrhages in the wall of the intestine, the intestine remains viable and is impermeable to potassium ferrocyanide. Higher pressure (20 cm. of water) is, however, tolerated for only ten to twenty-two hours and results in congestion of the intestine. When this degree of pressure is sustained over a longer period (twenty-four hours) necrosis occurs and permeation of the necrotic areas by the test substances can be demonstrated. Still higher pressures (40 cm. of water or more maintained for eleven hours) result in hemorrhage, necrosis and loss of viability with permeation of the wall of the bowel. However, such pathologic changes are not regularly observed at autopsy after experimental simple ileal obstruction. This emphasizes the importance of the ability of the hollow muscular viscera, such as the bowel and the bladder, to accommodate themselves to alterations of volume without significant changes in the intraenteric tension. Undoubtedly the intestine is able to maintain, within certain limits, an adequate blood supply, despite the compression of the capillaries and of the small venules which follows the increase of intraenteric tension. This may be due to a dilation or stretching of the circular muscle fibers of the intestinal wall. Moreover the dilation of the intestine in experimental and clinical obstruction occurs so gradually that considerable distention may be present without a great increase of tension.

The influence of obstruction on the physical character of the wall of the intestine was investigated. The intestine was found to become shorter by as much as 33 per cent of its initial length, and increased by more than 100 per cent in weight. When allowance has been made for the decrease in length, the actual increase in the weight of the obstructed bowel was found to be 34 per cent. Its water content was found to be increased only 7 per cent, and the amount of blood in the wall of the intestine was increased from one to ten times the normal amount. The tensile strength of the small bowel which has been subjected to simple obstruction was found to be considerably reduced when compared with that of the normal bowel.

The effect of distention on intestinal tone and peristalsis has been discussed. It has been shown that increasing the pressure results in increase of peristalsis, which is most marked in the ileum at pressures between 15 mm. and 30 mm. of mercury. Sudden decompression of the distended intestine provokes violent peristaltic activity. Tone has been demonstrated to be more important than small changes of volume in affecting peristalsis and intraenteric pressure.

It has been well established by previous investigators that, while secretion is increased under conditions of obstruction, absorption is diminished, thus further augmenting the hydraulic factor which operates in a vicious cycle. In this study there was a marked decrease in absorption of all substances tested.

Under identical experimental conditions a closed loop of normal ileum absorbs in one hour approximately 90 per cent of the amount of water necessary to fill it, while a loop of ileum which has been subjected to simple ileal obstruction absorbs less than 10 per cent. The obstructed bowel reacts to increase of tension in exactly the same manner but to a lesser degree than does the normal bowel. The optimal pressure for absorption of water was found to be 40 cm. of water in both groups. There was a significant difference between the amount absorbed by the obstructed intestine and that absorbed by the normal intestine at pressures between 20 cm. and 60 cm. of water (fig. 5).

Lymphatic absorption of dyes and of bacteria is enhanced by the conditions of obstruction and by increased intraenteric pressure. In intestinal obstruction, especially of the closed loop type, dyes and bacteria have been demonstrated in the regional mesenteric lymph nodes. They could not be recovered from the chyle of the thoracic duct. These findings are in accord with those of Costain,[44] Murphy and Brooks,[45]

44. Costain, W. A.: Lymphaticostomy in Intestinal Obstruction, Surg., Gynec. & Obst. **38**:252-255, 1924.

45. Murphy, F. T., and Brooks, B.: Intestinal Obstruction: Experimental Study of the Causes of Symptoms and Death, Arch. Int. Med. **15**:392-412 (March) 1915.

and Stone and Firor,[5] who have emphasized the importance of the lymphatics as an avenue for absorption in obstruction. However, severance of the mesenteric lymph pedicle in cats with intestinal obstruction to prevent absorption of toxins through the lymphatics did not lengthen the period of survival. Thus, no evidence was adduced to indicate that the occurrence of increased lymphatic absorption is of much significance in the causation of death of animals with intestinal obstruction.

This work emphasized the significance of the mechanical factors in simple intestinal obstruction. The ill effects of obstruction can all be traced to the effect of distention on the wall of the bowel. The factor of loss of fluid, while important in high intestinal obstruction, has been shown by other investigators to be unrelated to the duration of survival in cases of low ileal obstruction. The main consideration appears to be the viability of the intestinal wall. The normal intestine prevents transperitoneal migration of bacteria and of substances which are toxic when absorbed through the peritoneal cavity. A sustained high intraenteric pressure results in impairment of the viability of the wall of the intestine and thus allows transperitoneal absorption. Relatively low pressures (20 to 40 cm. of water) maintained over a sufficiently long period produce structural changes in the intestinal wall and allow this process to take place.

In view of what has been said in the previous sections on the effect of distention in simple ileal obstruction on the function and the structure of the wall of the intestine, the sequence of events in the development of the changes described may be summarized in the following manner: With the onset of intestinal obstruction there is stasis of fluid and gas within the bowel. The stasis produces a slight increase of intraenteric pressure and a moderate degree of distention, which stimulate the normal bowel to increased peristalsis. Distention and increased intraenteric pressure result in an augmented secretion of intestinal juices, which adds to the content of the bowel. Absorption is decreased early in the course of the obstruction. There is thus a progressive increase of distention and of intraenteric pressure. With a rise in intraenteric pressure there is eventually manifested interference with the circulation of blood to the wall of the intestine. Venous stasis ensues and causes infiltration of the intestinal layers with leukocytes. Eventually hemorrhagic infarction, necrosis and even perforation of the intestine may take place. If the intraenteric pressure is maintained over a sufficiently long time, the viability of the wall of the bowel becomes impaired and its permeation by toxic material may take place through the gangrenous patches. Death is then due to peritonitis or to absorption of toxic material by way of the peritoneum. Relatively low pressures (20 to 40 cm. of water) if

maintained over a sufficiently long period (seventeen to thirty hours) to produce structural changes in the intestinal wall will allow this to take place.

The processes occurring in simple low ileal obstruction and in closed loop obstruction are listed here:

Obstruction
Intestinal stasis
Distention
Increased activity of the bowel
Increased intraenteric pressure
Increased secretion of intestinal juices
Decreased absorption
Further distention
Violent peristalsis
Circulatory stasis
Increased venous pressure
Anoxemia of the wall of the bowel
Hemorrhage
Impairment of viability
Leukocytic infiltration
Necrosis and gangrene
Loss of viability
Permeability of the wall of the bowel to toxins and bacteria
Peritonitis

INTESTINAL OBSTRUCTION

EXPERIMENTAL EVIDENCE ON THE LOSS OF BLOOD IN INTESTINAL STRANGULATION

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In the past, numerous attempts have been made to ascribe the disastrous effects of all types of intestinal obstruction to some one etiologic factor. Of the many explanations offered, that of intestinal toxemia has been given greatest consideration. Literally hundreds of investigators have tried to show that this or that toxin was the one responsible for the dire results. However, there has been no general agreement as to what toxin was to blame. This, together with the fact that no theory of intestinal toxemia has ever offered any benefit to the patient in the way of improved methods of treatment or lowered mortality, has cast grave doubt on this explanation. The work of the past thirty years, however, has shown that, from an anatomic and pathologic standpoint at least, intestinal obstructions can be divided into two major types or a combination of the two. The terms simple and strangulation obstruction have been used to designate and differentiate the two major forms by clinicians and investigators alike.

The first term, simple obstruction, implies an occlusion of the lumen of the bowel without gross interference with the mural blood supply, while the second, strangulation obstruction, implies a vascular impairment of the wall of the bowel and its mesentery. At operation or necropsy, patients for whom the diagnosis was made clinically not infrequently show some evidence of both types of obstruction. In fact, most strangulation obstructions are accompanied by a simple mechanical or neurogenic obstruction. The reverse is not usually the case. Occasionally, however, a simple obstruction may produce some intramural vascular changes, even to the point of necrosis and perforation of the intestinal wall. This is especially true when a so-called gas trap develops and produces stasis of blood within the wall of the bowel as a result of a pathologically high intraintestinal pressure. This occurs more frequently in the colon, where a neoplastic lesion of the sigmoid

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flexure may produce a closed loop. The usual competency of the ileocecal valve, which prevents the backward passage of gas into the small intestine, may convert such a loop into a gas trap.

Because of a rather frequent combination of simple and strangulating mechanisms, with their overlapping symptom complexes, many patients observed clinically fail to present typical symptoms of either type of obstruction. Therefore, it has been rather difficult for the surgeon and the clinician to realize that two entirely different physiopathologic processes might be responsible for producing the many complex effects of intestinal obstruction. As a result, the symptoms have been erroneously attributed to some one etiologic factor. It is no wonder that the theory of intestinal toxemia has become so firmly rooted in the consciousness of medical practitioners that the concepts advanced in recent years have been but slowly and hesitatingly accepted.

Recently several writers (Wilkie,[1] Murphy,[2] Elman,[3] Scott,[4] Blalock[5] and Mensing[6]) have called attention to the fact that the loss of blood in intestinal strangulations may be of some consequence in bringing about and aggravating symptoms of shock and in causing death. D. P. D. Wilkie,[1] writing in 1913, distinguished clearly between simple and strangulation obstruction. He was impressed with the amount of blood lost in strangulation obstruction and felt that it played an important role in the causation of shock and death. During the course of some experiments on strangulation obstruction in the dog, I also observed significant loss of blood into the walls and lumen of the strangulated bowel. Further experiments were devised to determine the extent of this loss of blood and its relation to changes in blood pressure, shock and death as seen in these conditions. The presentation of the results of these experiments constitutes the main object of this paper.

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1. Wilkie, D. P. D.: Experimental Observations on the Cause of Death in High Obstruction, Brit. M. J. **2**:1064, 1913.

2. Murphy, F. T., and Vincent, B.: An Experimental Study on the Cause of Death in Intestinal Obstruction, Boston M. & S. J. **165**:684, 1911.

3. Elman, R., and Cole, W. H.: Loss of Blood as a Factor in Death from Acute Portal Obstruction, Proc. Soc. Exper. Biol. & Med. **29**:1122, 1932.

4. Scott, H. G., and Wangensteen, O. H.: Blood Losses in Experimental Intestinal Strangulations and Their Relationship to Degree of Shock and Death, Proc. Soc. Exper. Biol. & Med. **29**:749, 1932.

5. Blalock, A.: Trauma to the Intestines: The Importance of the Local Loss of Fluid in the Production of Low Blood Pressure, Arch. Surg. **22**:314 (Feb.) 1931.

6. Mensing, E. H.: Intestinal Obstruction: Application of Newer Principles Evolved from Experimental and Clinical Experience, Am. J. Surg. **17**:206, 1932.

METHOD OF EXPERIMENTS

In order to evaluate the role that the arteries and veins may play individually and collectively in determining the period of survival of dogs with strangulation obstruction, the experiments here reported were divided into four groups according to the type of strangulation produced. Variations in the length of intestine obstructed also were taken into account. In the creation of the various types of obstruction described in this paper, ¼ to ½ grain (16 to 32 mg.) of morphine sulfate and 1/150 to 1/75 grain (0.4 to 0.9 mg.) of atropine sulfate were given preoperatively to each animal. Ether was used as the anesthetic in every instance, and sterile technic was employed throughout.

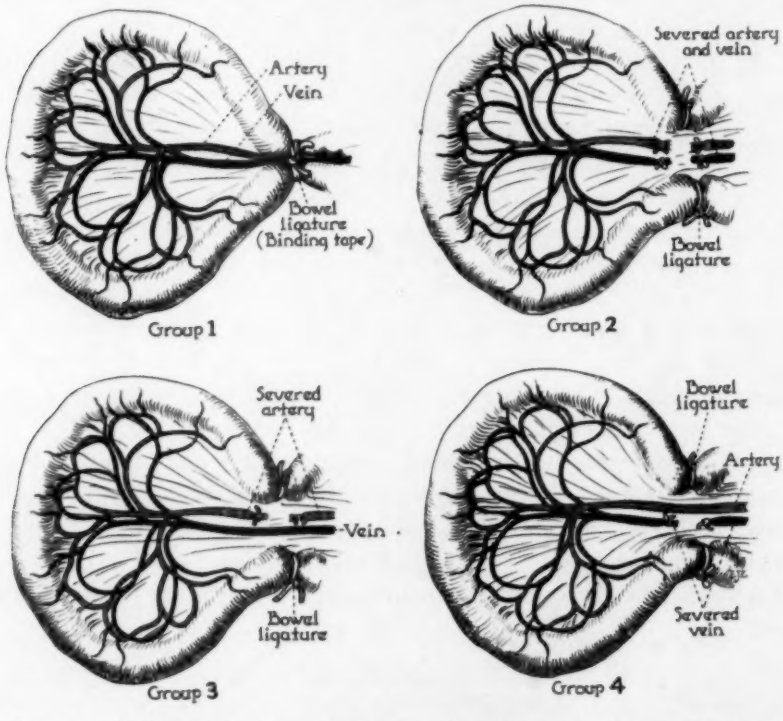

Fig. 1.—The four methods employed in producing the experimental strangulation obstructions recorded in this paper. Group 1 presents a partial occlusion of lumen and blood supply; group 2, a complete occlusion of blood supply; group 3, a complete arterial occlusion with patent vein, and group 4, a complete venous occlusion with artery intact.

Type of Obstruction.—In group 1 no attempt was made to control the relative degree of arterial or venous occlusion of the strangulated intestinal loops. In this way it was hoped that some idea might be obtained of the variations in length of life which one might encounter experimentally when a constricting band was passed about the mesenteric pedicle and the wall of the bowel. Binding tape, 1 cm. in width, was passed about the mesenteric vessels and then carried around the wall of the bowel and tied. The lumen of the bowel as well as its blood supply was thus occluded. The method is illustrated in figure 1. An experimental volvulus,

or adhesive band type of strangulation, was thus brought about, which simulated closely the variations in this type of obstruction usually met in clinical experience.

In groups 2, 3 and 4, binding tape was used merely to occlude the lumen of the bowel at the ends of the segment strangulated. A window was first made in the mesentery adjacent to the wall of the bowel at the ends of the loop selected. The anastomosing vessels adjacent and parallel to the wall of the bowel were cut and ligated. The binding tape was passed about the wall of the bowel and tied. The mesenteric vessels were then occluded by ligating and severing them individually or collectively, according to the type of obstruction desired. In group 2, all the arteries and veins to the loop selected were ligated and severed. In group 3, all the arteries were ligated, but the veins were left intact. In group 4, all the veins were ligated but the arteries were left intact. Figure 1 illustrates the four different types of obstruction produced. After operation the animals were returned to their cages or kept on the operating table while their blood pressure was being recorded on the kymograph. No particular postoperative treatment was given. Most of the experiments being of an acute nature, problems of feeding and hydration did not have to be considered. Immediately after death the animals were subjected to careful autopsy.

RESULTS

Pathologic Observations at Autopsy.—At autopsy a marked difference was noted in the appearance of the strangulated bowels in the various groups. In group 1 (obstruction by encirclement), the peritoneal cavity was found to contain from 30 to 140 cc. of bloody serous fluid. The hemoglobin content of this fluid was found to vary between 3 and 10 per cent. The bowel itself was always moderately distended. The color was usually a deep purple or dark mahogany. The degree of distention apparently varied with the degree of tension on the mesenteric pedicle. The greater the tension the less the distention, apparently because the arterial blood was prevented from entering the wall and lumen of the bowel. The serosa in most cases was found to have a fibrinous exudate over its surface, although in some few instances little or no fibrin was present. When the bowel was sectioned, the wall was seen to be definitely thickened. The lumen was observed to contain a moderate amount of dark bloody fluid or actual blood clots. The hemoglobin content of this fluid was found to vary between 60 and 140 per cent. On microscopic as well as gross section, the wall of the bowel was observed to be markedly thickened in comparison with its normal condition. The reason for this increase was apparent on examination. The mucosa, especially, was seen to be packed with red blood cells. There were hemorrhages into the muscular layers as well.

In group 2 (complete arteriovenous obstruction), the peritoneal cavity was found to contain 40 to 350 cc. of a dark, foul-smelling bloody fluid when the strangulated loops showed gross rupture. When the loops remained intact the peritoneal cavity contained less fluid, which was less hemorrhagic and not nearly so foul smelling. The hemoglobin

content of this fluid was usually less than 10 per cent. Examination of the bowel revealed it to be lusterless, dark purple or mahogany colored and moderately distended with bloody fluid when not ruptured. In this group and in the next (arterial obstruction), in which the pathologic findings were practically identical, 18 of 28 animals had loops which were ruptured in one or more places at the time of death. The wall of the intestine in these animals was usually thinner than normal, extremely friable and jelly-like. There were a few exceptions to these general statements. On 3 or 4 occasions, the intestine was found to be a patchy grayish yellow green; the wall was not perforated and the lumen not distended. This type of infarction was termed anemic

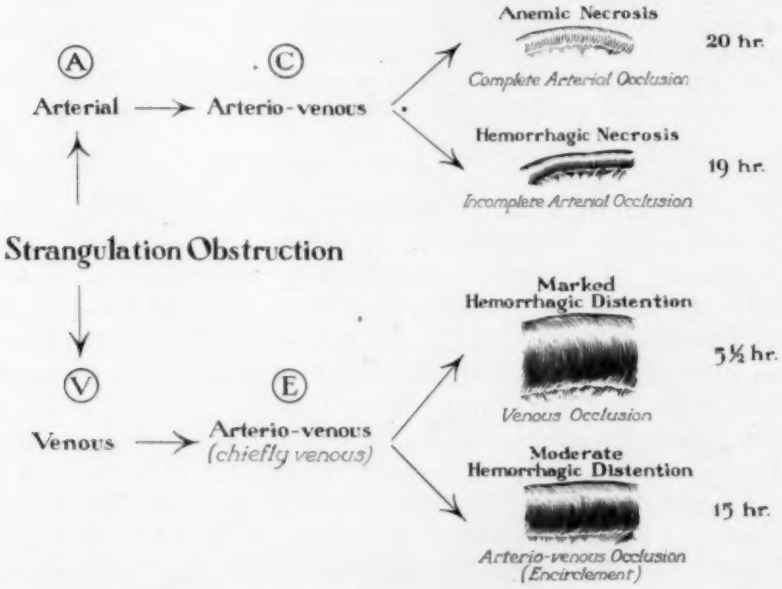

Fig. 2.—The various types of strangulation obstruction that may result, according to the relative degree of venous or arterial occlusion. When the arterial obstruction predominates there is little or no distention, whereas when the venous obstruction predominates there is a moderate to marked distention with blood.

necrosis, in contrast to the usual hemorrhagic necrosis seen in other types of strangulation. A careful analysis of the methods employed revealed the cause for the hemorrhagic necrosis in some instances and the anemic necrosis in others. In the cases of hemorrhagic necrosis blood apparently gained access to the wall of the bowel through the capillary bed of the mesentery proper, whereas no blood at all entered the bowel in the cases of anemic necrosis.

In group 3 (arterial obstruction), the pathologic picture, as previously stated, was practically the same as in group 2. In group 4, the

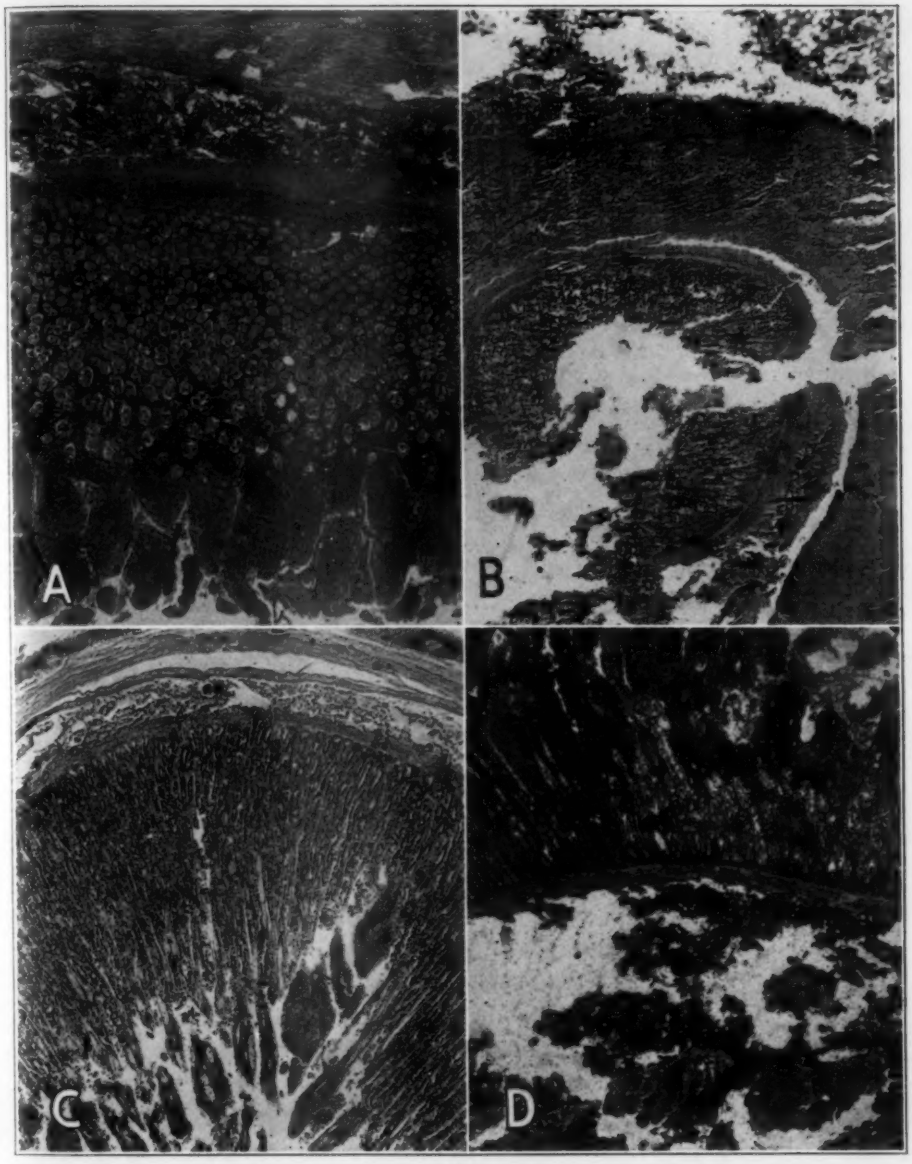

Fig. 3.—*A*, photomicrograph of an ileum after an encirclement ligature had been tied about the wall of the bowel and the mesentery for two hours. Note the thickening of the mucosa and submucosa, the villi being packed with blood cells. Note the blood in the submucosa and the hemorrhages in the muscular layers. *B*, photomicrograph of an ileum in which the arteries and veins had been completely severed for fifteen hours. This is grossly a case of hemorrhagic necrosis. Note the sloughing mucosa and serosa and the absence of any definite muscular layers. *C*, photomicrograph of an ileum in which the arteries and veins had been completely severed for thirty hours. Note the essentially normal histologic picture, except for the general atrophy of all layers of cells and the slight amount of blood in the tips of the villi. *D*, photomicrograph of an ileum in which the veins had been completely ligated for four hours. Note the massive hemorrhages into all layers. This is grossly a case of marked hemorrhagic distention. Note how the muscle bundles are torn apart. Only the serosa and the basement layer of mucosa remain intact in this section.

pathologic changes were almost identical with those in group 1, except that they were all more marked. The peritoneal fluid was present in larger amounts, the quantity ranging between 30 and 300 cc. The degree of distention of the bowel was likewise more marked. When the bowel was opened it was seen to contain rather large amounts of bloody fluid with a hemoglobin content ranging between 90 and 140 per cent (Sahli).

Increase in Weight of Strangulated Loops of Intestine.—When the strangulated bowels in groups 1 and 4 were weighed after the conclusion of the experiments, they were noted to have increased in weight over estimates based on a series of normal weights. The increase was from 133 to 235 per cent in group 1 and from 150 to 490 per cent in group 4. This increase was due solely to blood cells and plasma which had accu-

TABLE 1.—*Percentage of Increase in Weight of Strangulated Loops of Ileum in Eighteen Dogs*

	Weight of Dog, Kg.	Length of Intestine, Ft.	Weight of Intestine, Gm.	Normal Weight, Gm.	Percentage of Increase in Weight
Group 1 (encirclement of pedicle)					
Minimum	11	2.0	240	76	133
Maximum	21	5.0	675	150	320
Average	15	3.2	460	103	235
Group 4 (ligation of veins)					
Minimum	8	3.0	260	90	150
Maximum	22	5.5	1,030	170	490
Average	15	4.3	680	135	283

mulated within the lumen and the wall of the intestine during the period of strangulation. Table 1 gives the minimum, maximum and average weights of strangulated bowels obtained at autopsy from a series of 18 dogs, 7 being in group 4 and 11 in group 1. The figures for normal weights of intestines are estimates based on tabulations of weights of given lengths of small intestine taken from 10 normal dogs. The average weight for 1 foot (30 cm.) of normal ileum was found to be approximately 30 Gm. and 1 foot (30 cm.) of normal jejunum 35 Gm. Table 1 shows in percentage the increase in weights of strangulated loops of ileum in 18 dogs.

Total Protein Content of Peritoneal Fluid.—From the gross pathologic condition of the bowels, the microscopic picture and the increase in weight of the intestine, it was evident that a rather large amount of blood was lost into the lumen and the wall of the bowel. This was especially true in the group with patent arteries and ligated veins. The peritoneal cavity, as already noted, was observed to contain a moderately large amount of clear blood-tinged fluid. Calculations of total protein content of the peritoneal fluid were made in three of the groups,

those in which venous, complete and arterial ligation, respectively, were performed. The group in which the pedicle was encircled was omitted, because it was felt that any differences which might be found in this group would lie well within the extremes of the group with venous ligation, on the one hand, and that with arterial or complete ligation, on the other. Table 2 gives the data in 17 experiments in which the total protein content of the peritoneal fluid and blood plasma was determined at operation or at the time of the animal's death. The total protein content of the blood plasma was determined on samples of blood drawn at the time of the removal of the peritoneal fluid. It will be noted that when the loop of bowel was not ruptured the total protein content of the peritoneal fluid was practically the same as that of the animal's own blood plasma. When the loop was ruptured, the protein content rose to as high as twice that of the blood plasma. It will be

TABLE 2.—*Total Protein Content of Peritoneal Fluid in Twenty-One Dogs with Strangulated Loops of Ileum*

	No. of Dogs	Type of Obstruction	Average Length of Strangulated Bowel, Ft.	Average Time of Strangulation, Hr.	Average Total Protein Content of Blood	Average Total Protein Content of Peritoneal Fluid
Group 2	10	Arterial and venous	2.2	26.6	5.38	6.47
Group 3	6	Arterial	3.0	11.3	5.62	6.32
Group 4	5	Venous	3.8	4.1	5.10	4.69

seen that in the group with venous ligation, at least, the peritoneal fluid is not unlike the animal's own blood plasma, having a practically equivalent total protein content.

Effect of Intravenous Injections of Peritoneal Fluid.—In order to determine whether or not the peritoneal fluid of animals suffering from the various types of strangulation obstruction was toxic, portions of it were collected at operation or autopsy and introduced into the veins of the legs of normal dogs. The amounts varied between 20 and 185 cc. No attempt was made to filter, heat or treat this fluid in any way. It was collected under aseptic technic and introduced with a sterile needle and syringe. At the time the fluid was introduced into the vein of the leg a tracing of carotid blood pressure was made with the animal under light ether anesthesia. Fifteen experiments were carried out on 14 different dogs. There were no immediate or delayed effects in 10 of the animals. These 10 animals had been given peritoneal fluid obtained from animals that died without gross rupture of the strangulated loops. In the 4 remaining animals there was an immediate effect which consisted of a marked sudden fall in blood pressure followed by a sharp rise. By the next day all 4 animals had died of generalized gas bacillus

infection. Three of these 4 had received peritoneal fluid obtained from animals in which the loop had ruptured prior to death. The fourth animal from which peritoneal fluid had been taken died with an intact loop, but the wall of the intestine was necrotic. The peritoneal fluid obtained from this animal, as well as that from the 3 with ruptured loops, had a foul odor, and bacteriologic studies revealed the presence of innumerable bacteria, both anaerobic and aerobic.

In the 10 animals that showed no immediate effect other than a slight rise in blood pressure there was likewise no delayed effect. Within an hour or two after the discontinuance of the anesthesia, the animals

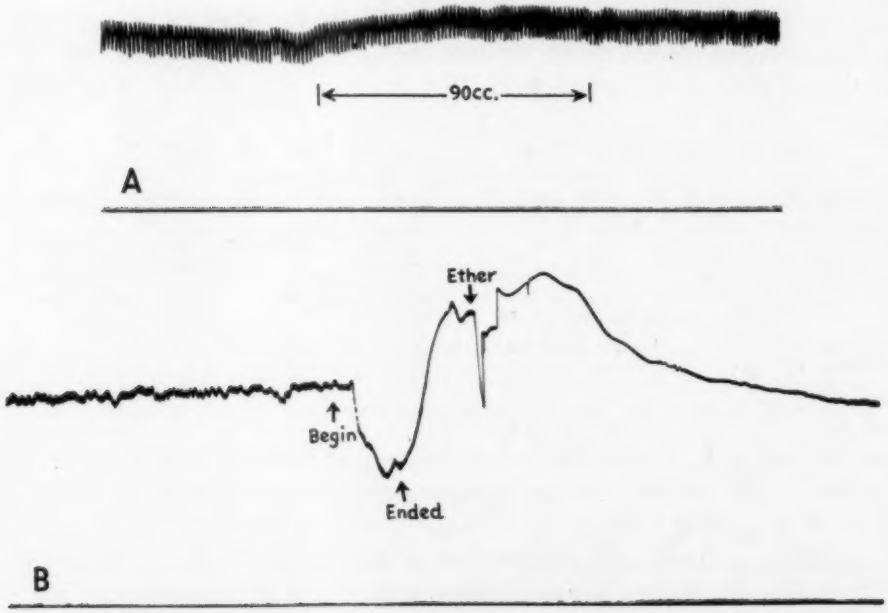

Fig. 4.—*A*, effect on the carotid blood pressure of a normal dog of injecting intravenously 90 cc. of peritoneal fluid from a dog with a strangulation obstruction (encirclement) of eighteen hours' duration. The strangulated loop of bowel was not grossly ruptured. The recipient suffered no immediate or delayed effect. *B*, effect on the carotid blood pressure of a normal dog of injecting intravenously 40 cc. of peritoneal fluid from a dog with a strangulation obstruction (ligation of arteries and veins) of fifteen hours' duration. The strangulated loop was grossly ruptured. The recipient died by the following morning.

were up and about as usual. The next day they ate well and appeared normal in all respects. The tracings shown in figure 4 are typical of the effects which have been described. From these experiments it may be concluded that the peritoneal fluid when injected intravenously has no appreciable effect on the blood pressure or general health of the

animal unless the strangulated loop is ruptured or is about to rupture. When death occurs it is apparently due to the intravenous injection of bacteria.

Absorption of Strychnine Sulfate From the Normal Bowel and From the Strangulated Bowel.—In view of the controversy concerning the absorption of so-called toxic products from the obstructed bowel, it was deemed wise to test the absorptive power of the normal and of

TABLE 3.—*Effect of Intravenous Injection into Normal Dogs of Peritoneal Fluid from Dogs With Strangulation Obstruction*

Experiment	Duration of Strangulation, Hr.	Condition of Strangulated Bowel	Amount of Injection, Cc.	Effect of Injection on Normal Dogs
33 E*	4	Intact	100	No immediate or delayed reaction in 2 dogs
34 E	5½	Intact	50	No immediate or delayed reaction
36 E	6	Intact	50+ 90 blood serum	No immediate or delayed reaction other than slight (10 mm.) rise and fall in blood pressure
41 E	8½	Intact	185	No immediate or delayed reaction other than slight rise in blood pressure
37 E	6½	Intact	80 + 45	No immediate or delayed reaction other than slight rise in blood pressure
42 E	10	Intact	120	No immediate or delayed reaction other than slight rise in blood pressure
43 E	18	Intact	90	No immediate or delayed reaction other than slight rise in blood pressure
44 E	17	Ruptured	100	Abrupt fall in blood pressure followed by rise, death within 24 hr.
50 V	1	Intact	20	No effect
47 C	7	Intact	20	No effect
45 C	18	Intact, necrotic	20	Abrupt fall in blood pressure to 60 mm. followed by abrupt rise to 135 mm., death within 24 hr.
48 C	15	Ruptured	40	Very abrupt fall in blood pressure to 85 mm. followed by a rapid rise to 240 mm., death within 24 hr.
46 C	27	Ruptured	20	Marked fall in blood pressure followed by gradual rise, death within 24 hr.

* E, encirclement of pedicle; V, ligation of veins; C, ligation of veins and arteries.

the strangulated intestine for products of known toxicity. Strychnine sulfate was chosen for this purpose, as it produces unmistakable clinical symptoms when absorbed in small quantities. A 50 mg. dose of strychnine sulfate was introduced into the lumen of the normal small intestine in 3 dogs. A similar dose was placed free in the peritoneal cavity in 2 other dogs. All 5 animals showed definite signs of irritability, followed by convulsions in from three to five minutes, and subsequently died. Fifty to 150 mg. of strychnine sulfate was then introduced into the lumens of strangulated loops of bowel immediately after strangulation. The usual four types of strangulation were employed.

In group 1 (by strangulation encirclement), 3 experiments were carried out. One hundred and fifty milligram doses were employed. In all 3 animals convulsions were delayed. In the first animal convulsions developed in about four hours, and death ensued in four and one-half hours. The second and third animals died in convulsions after seven and fifteen hours respectively. The first signs of irritability or convulsions appeared just a few hours prior to death. These results indicate a great delay in absorption as compared with the normal rate. The wide variation in time of development of symptoms was to be expected in this group in view of the fact that no attempt was made to arrest the flow of blood completely, the degree of occlusion depending entirely on the tightness of the encircling tape. The peritoneal fluid was tested for the presence of strychnine after the death of the animal by the injection of 2 to 5 cc. samples of it into the dorsal lymph sacs of frogs, which were then observed for tetanic convulsions. None of the frogs gave a positive reaction, except that in which the sample obtained from the animal that died after fifteen hours had been injected. This animal had a grossly perforated loop, and it was to be expected that its peritoneal fluid would contain strychnine.

In group 2 (complete strangulation), 10 experiments were carried out. Six animals showed no sign of convulsions, while delayed convulsions developed in 4. Death occurred in these animals after six, seven, twelve and fourteen hours respectively, and convulsions were first observed in each instance only an hour or two before death. When the peritoneal fluid from these 4 animals was tested by injection into frogs, the presence of strychnine was indicated only in the postmortem specimen from the animal that died after twelve hours. Of the 6 animals that died without tetany or convulsions, the same test indicated the presence of strychnine in the postmortem specimen from only 1, which had lived thirty hours after strangulation. The strangulated loop in this animal was extremely necrotic and friable but not grossly perforated. In group 3 (arterial obstruction), 10 experiments were carried out. None of the 10 animals showed signs of strychnine poisoning. There was some evidence of minute amounts of strychnine in the peritoneal fluid, as convulsions developed in from twelve to forty-five minutes in 3 frogs into which it was injected. In group 4 (venous obstruction), 9 experiments were carried out. Convulsions developed in 3 of the 9 animals. The explanation for these results was evident at autopsy, when the normal adjacent intestine was seen on being opened to contain bloody fluid from the strangulated loop.

Although these experiments might seem to indicate that toxic products can be absorbed from the strangulated bowel, one must remember that when convulsions did occur they were greatly delayed. Moreover, as pointed out in reference to the last group, the possible avenue of

absorption from the adjacent normal bowel must not be overlooked. While strychnine is readily absorbed from the normal bowel, as the control experiments showed, numerous workers have failed to find any evidence of absorption of "toxins" contained in strangulated loops from normal bowel. However, there is no denying that traces of strychnine probably pass through the wall of the bowel the nutrition of which has been seriously impaired, since in a number of cases its presence in the peritoneal fluid was indicated by the reactions of frogs into which the fluid was injected.

Absorption of Histamine and Tetanus Toxin from the Strangulated Bowel.—It has been noted occasionally that after the release of a strangulating mechanism a patient previously in good condition immediately goes into a state of shock. This fact has been used as an argument for the absorption of a toxic, histamine-like substance. Blalock,[5] however, has recently shown that under such circumstances blood and plasma are lost through the traumatized vessels into the tissue spaces, where they are removed from the general circulation, and a deficit in the volume of blood returning is thus caused. Wangensteen and Loucks [7] could not confirm the absorption of histamine, even though they introduced it into the lumens of strangulated loops. On releasing the strangulating mechanism, they noted not infrequently a fall in blood pressure and sometimes even death. However, they never noted the characteristic drop in blood pressure that one would expect to accompany the absorption of histamine.

These experiments were repeated, with relatively large amounts of histamine, and the authors' results were confirmed. No effect of histamine was noted in any of the tracings of blood pressure which were made. In fact, in 4 animals subjected to a four hour strangulation by encirclement, release of the strangulating ligature after the introduction of 75 to 150 mg. doses of histamine into the strangulated loops caused only a slight gradual decline in the carotid blood pressure in a single instance. Fifty milligrams of tetanus toxin likewise was introduced into the lumen of a completely devascularized loop of bowel in 1 instance. Samples of peritoneal fluid were obtained by aspiration through an intraperitoneal catheter at two hour intervals. These samples (3 to 5 cc.) were injected subcutaneously into white mice. Six mice received injections over a period of twelve hours. They were observed for forty-eight hours, and none showed evidence of tetanic seizures. These experiments indicate that histamine or a histamine-like substance is not absorbed from a previously strangulated bowel. In fact,

7. Wangensteen, O. H., and Loucks, M.: Studies in Intestinal Obstruction: II. The Absorption of Histamine from the Obstructed Bowel, Arch. Surg. **16:** 1089 (May) 1928.

all the evidence in this and the preceding section would point to a delay rather than an acceleration in absorption from the damaged wall of the bowel. Furthermore, the devitalized wall of the bowel is apparently impermeable to tetanus toxin, at least over a twelve hour period.

Intraperitoneal Autolysis of Autoclaved Intestine and Intestine Treated with Chemical Antiseptics.—In order to determine whether the wall of the bowel itself is "toxic," an attempt was made to free the normal intestine of the dog from its usual bacterial flora by chemical and thermal sterilization. Segments of bowel thus treated were placed in the peritoneal cavities of normal dogs, in order to determine their relative and actual degree of "toxicity," as judged from a clinicoexperimental standpoint. In every instance in which segments of intestine were treated with chemical antiseptics for periods varying from five to ten minutes, cultures of mucosal scrapings were observed to contain a great variety of organisms. When these chemically treated loops were

TABLE 4.—*Resulting Mortality in Twenty-Eight Dogs When Segments of Small Intestine Obtained from Other Dogs Were Treated in Various Ways and Introduced into the Peritoneal Cavity*

Number of Experiments	Method	Percentage of Mortality
2	Open loops of ileum, devascularized	100
3	Open loops of jejunum, devascularized	100
2	Devascularized Thiry-Vella loops	100
4	Segments of bowel treated with tincture of iodine	100
4	Segments of bowel treated with mercurochrome and alcohol	75
3	Segments of bowel treated with water and ether	100
10	Segments of bowel autoclaved 30 to 45 minutes	10

placed in the peritoneal cavities of 18 normal animals, only 1 survived. In contrast to this, 9 of 10 animals survived that had loops of bowel sterilized by steam placed in their peritoneal cavities. Cultures of the mucosa from the autoclaved loops were negative in all instances. A few organisms must have been present in the mucosa of the bowel in 1 case or have gained entrance at the time of operation, as the animal died five days later of bacterial peritonitis. These results were to be expected in view of the fact that the devitalized intestine and the peritoneal cavity provide bacteria with an ideal culture medium under optimum temperatures. This work substantiates the experimental results of Dragstedt [8] and his co-workers, who showed that it was extremely difficult to sterilize the normal bowel with the usual antiseptics. It also confirms their conclusion that devitalized sterile loops of bowel are harmless.

8. Dragstedt, L. R.; Dragstedt, C. A.; McClintock, J. T., and Chase, C. D.: Intestinal Obstruction: II. A Study of the Factors Involved in Production and Absorption of Toxic Materials from the Intestine, J. Exper. Med. **30**:109, 1919. Dragstedt, L. R.; Moorehead, J. J., and Burcky, F. W.: Intestinal Obstruction: I. An Experimental Study of the Intoxication in Closed Intestinal Loops, ibid. **25**:421, 1917.

Bacteriologic Observations of the Peritoneal Fluid After Strangulation Obstruction.—Thirty-five centimeter loops of the lower part of the ileum were strangulated for periods varying from two and three-quarters to four and one-half hours with an encircling ligature. Bacteriologic studies were then conducted as follows: As strict aseptic technic as possible was employed at the time of strangulating and taking of cultures. In each study smears were made of the peritoneal fluid, the wall of the bowel and the mesentery. Cultures were made on lactose, liver peptone and brain mediums. Smears were also made of cultures twenty-four and forty-eight hours later. The following organisms were found:

1. Gram-positive spore-forming rods
2. Gram-positive micrococci
3. Gram-positive diplococci
4. Gram-negative rods
5. Clostridium welchii
6. Bacillus coli
7. Aerobacter aerogenes
8. Streptococcus haemolyticus
9. Sarcina
10. Staphylococci

In 10 experiments there were 6 positive and 4 negative cultures. There were likewise 4 deaths. Three of the deaths occurred within two hours of release and were undoubtedly due to hemorrhage, as will be shown in the succeeding sections. One of these deaths was that of an animal which had entirely negative cultures. Three of those that recovered (50 per cent) had positive cultures. Therefore, the presence of organisms was not as serious a factor in the causation of death as it might seem on the face of it. This fact is mentioned in order to show how much the bacteriologic aspects of intestinal obstruction have been overemphasized.

Fall in Hemoglobin Content After Venous Obstruction.—A definite fall in hemoglobin content was noted in all animals with venous obstruction. The results obtained in 1 animal that died and 2 in which resection of the bowel was carried out are shown in table 5.

Correlation Between the Fall in Blood Pressure and the Time, Length and Type of Intestinal Strangulation.—The carotid artery was cannulated in 30 dogs and a tracing made of the blood pressure during the course of vascular occlusions of various types. In each experiment the time as well as the length and site of strangulation of the bowel was noted. In order to control the degree of arterial and venous occlusion, the experiments were divided into four groups, as defined in the preceding sections. In group 1 (obstruction by encirclement), the length of bowel strangulated varied between 1 and 11½ feet (30 to 350 cm.). The time varied between three and seven hours. The blood pressure prior to strangulation was recorded in 5 of these experiments, while the resulting pressure after from three to seven hours was ascertained in every case. The lowest pressure recorded after this interval was 77 mm.

of mercury and the highest 140 mm. In 7 dogs, in which the length of intestine was less than 3 feet (90 cm.), the blood pressure had not fallen in any case below 100 mm. In the 9 remaining dogs, in which the segment strangulated was more than 3 feet (90 cm.) in length, the pressure at the time of release was less than 100 mm. in 6 instances. The time at which the release was effected varied within each division of this group, but the interval was approximately the same in both divisions, with but 1 exception. In group 2, in which the arteries and veins were ligated, the pressure was found to be within normal limits after from four to six hours' observation. Also in group 3, in which the arteries were ligated, the pressure was found to be within normal limits after from four to six hours. Tracings were taken after eighteen and one-half hours in the case of 2 animals which appeared moribund at that time. One showed a mean pressure of 20 mm. and the other of

TABLE 5.—*Decline in Hemoglobin Content of the Blood of Three Dogs in Which Strangulation Obstruction of the Small Bowel with Ligation of the Veins Was Produced*

Dog 303		Dog 304		Dog 305	
Time	Percentage of Hemoglobin	Time	Percentage of Hemoglobin	Time	Percentage of Hemoglobin
8:30 a.m.	108	8:15 a.m.	106	10:00 a.m.	95
9:10	Strangulation	9:50	Strangulation	10:30	Strangulation
9:40	103	10:20	96	11:00	84
10:40	105	11:20	102	12:00 noon	70
11:40	102	12:20 p.m.	78	12:50 p.m.	68
12:50 p.m.	90	1:20	65	1:50	65
1:50	82	2:10	Resection	1:40	Resection
2:50	76	2:20	68	2:50	62
3:35	Dog dead	3:25	70	3:50	68

70 mm. The first died half an hour later and the second one and one-half hours later; these observations signify that, though it was delayed, there was a definite fall in blood pressure terminally. In group 4, consisting of 10 dogs in which the veins were ligated, the pressure was below 70 mm. in every instance after from one to five hours. Three of the 10 animals were dead within this time. One animal, in which the superior mesenteric vein was ligated, died within one hour. Two of 3 dogs in which the veins to a 5½ foot (170 cm.) segment were ligated died within four hours, with consistent decline in blood pressure during the course of the experiments. One animal in which the veins to a 5 foot (150 cm.) segment were occluded was still alive at the end of four hours but had a blood pressure of only 54 mm. The remaining 6 dogs, in which veins were occluded to a segment of less than 3 feet (90 cm.), were all alive after an interval varying from three to five and one-half hours.

In several instances the pressure within the mesenteric veins to the loop of intestine selected was measured while a venous obstruction

was being produced. In each instance the pressure was seen to rise rapidly and quickly level off only slightly below the systemic arterial pressure. The fact that the venous pressure may rise to heights almost equivalent to the arterial pressure probably accounts for the rupture of the capillary bed of the wall of the bowel. There appears to be a direct correlation between the fall in blood pressure,

TABLE 6.—*Changes in Blood Pressure Produced by Various Types of Strangulation Obstruction of the Small Intestine*

Experiment No.	Weight of Dog Kg.	Length of Intestine Obstructed, Ft.	Time of Strangulation, Hr.	Initial Blood Pressure, Mm.	Resulting Blood Pressure, Mm.
		Group 1: Encirclement of Pedicle			
5	20	1	4	...	140
14	5	1	5	...	140
1	11.3	1	3	...	100
2	16	1	4.5	...	130
3	9	1	5	...	100
8	11	3	4.25	...	130
7	20	3	4	...	110
47	8	4	5	110	90
9	19	5	4.5	130	100
11	10	5	3.75	...	94
13	8	4	4.5	...	110
32	..	4	5	...	90
13	10	5	4	...	90
10	5.5	5	4.5	...	80
31	..	7	7	...	120
36	25.5	11.5	4.5	...	70
		Group 2: Ligation of Arteries and Veins			
17	20	4.5	4.75	...	120
16	14	5	4	...	145
16	14	5	6	...	135
115	18	2.5	18.5	...	20
115	18	2.5	19	...	Dead
116	22	2	18.5	...	70
116	22	2	20	...	Dead
		Group 4: Ligation of Veins			
101	13.5	2	2	120	56
100	20	2.5	4	150	60
93	9	2.5	4.5	120	60
99	19	2.5	5.5	140	70
91	21	2.5	5	140	70
92	12	3	3	110	38
55	20	5	4	140	Dead
56	16.2	5	4	150	56
51	13	5.5	2.5	120	Dead
114	10	..*	1	140	Dead

* In this animal the superior mesentary vein was ligated.

on the one hand, and the time, length and type of vascular occlusion, on the other. In general, the longer the time and the longer the loop strangulated, the greater the fall in blood pressure. The type of obstruction, however, is of even greater importance. In those instances in which the veins alone were ligated and in the strangulations involving encirclement (group 1), in which the arteries were not entirely occluded, the fall in pressure was much more rapid than in those instances (groups 2 and 3) in which the arteries were absolutely occluded, with or without occlusion of the veins.

Period of Survival After Various Types of Obstruction.—The four usual types of obstruction were produced in a number of instances with a view to determining the average length of life in the various groups. The lengths of bowel strangulated were varied in order to determine the variations which might result in accordance with the observations of Foster and Hausler.[9] In the experiments of group 1, in which an encircling ligature was placed about the mesentery and bowel in 14 dogs, the period of survival varied between four and twenty-eight hours. The mean length of life was sixteen hours. The length of bowel strangulated varied between 1 and 5 feet (30 to 150 cm.). The dog with the shortest obstruction lived the longest, an observation which is in accordance with Foster and Hausler's views. However, the dog with the longest obstruction lived longer than a number of other dogs with shorter obstructions. This and other exceptions to Foster and Hausler's conclusions were anticipated in view of the fact that the relative degree of arterial and venous obstruction could not be accurately controlled in this group. Further, the animals varied in weight as well (10 to 25 Kg.). Therefore, the actual length of intestine obstructed gives no indication of the relative amount obstructed, which is actually of greater significance. In 18 experiments of group 2, in which the arteries and veins were completely ligated, the shortest period of survival was eleven hours and the longest thirty-two. The length of intestine strangulated varied as in the preceding group between 1 and 5 feet (30 to 150 cm.). The mean length of life for this group was nineteen hours. In group 3, in which the arteries were tied in 9 dogs, the shortest period of survival was fifteen hours and the longest twenty-four hours. The length of bowel strangulated varied between 1 and 4 feet (30 to 120 cm.). The mean length of life was twenty hours. In group 4, in which the veins were tied in 15 animals, the shortest period of survival was two and one-half hours. The length of intestine varied between 3 and 5½ feet (90 to 170 cm.). The mean length of life was five and one-half hours.

Examination of table 7 reveals that the length of life in group 4, in which venous obstruction was produced was approximately one-fourth that in group 3, in which the arteries were obstructed, and in group 2, in which combined ligature of arteries and veins was performed; it was approximately one-third that of group 1, in which the degree of arterial occlusion was not absolute in all cases. Although the length of bowel involved is of great importance in shortening or prolonging the period of survival after strangulation obstructions, the type of obstruction seems to be of even greater significance, as a pure venous obstruction causes death in one-fourth the time that an arterial obstruction does.

9. Foster, W. C., and Hausler, R. W.: Studies in Acute Intestinal Obstruction: II. Acute Strangulation, Arch. Int. Med. **34**:697 (Nov.) 1924.

Loss of Blood in the Various Types of Intestinal Strangulation.—The data presented here on losses in the blood volume were obtained from experiments on 38 animals. As in the other phases of this work, the usual four types of intestinal strangulation were employed. At necropsy, in each instance, the peritoneal fluid was measured, the strangulated loop with its contents weighed, the contents removed and the intestine reweighed. The length of the strangulated bowel was measured just prior to the strangulation and in some instances afterward as well. The amount of blood lost was calculated as being equivalent

TABLE 7.—*Period of Survival After Strangulation Obstruction of the Small Intestine in Dogs*

	Number of Experiments	Length of Strangulated Intestine, Ft.	Average Period of Survival, Hr.
Group 1 (encirclement of pedicle)	1	1	28
	1	2½	18
	4	3	10
	1	3½	18
	5	4	14
	1	4½	18
	1	5	18
Group 2 (ligation of arteries and veins)	4	1	23½
	6	2	21
	3	3	18
	3	4	16
	2	5	13
Group 3 (ligation of arteries)	1	1	18
	3	2	21
	1	2½	18½
	3	3	22
	1	4	15
Group 4 (ligation of veins)	5	3	7½
	1	3½	3½
	1	4	4
	2	4½	4¾
	5	5	6½
	1	5½	2½

to the peritoneal fluid plus the increase in the weight of the intestine over the calculated normal weight. That these figures give an estimate of the true loss of blood in groups 1 and 4 is evident from the results described in the preceding paragraphs. The loss in the blood volume was then calculated from these figures, the total blood volume being based on the arbitrary figure of 7.5 per cent of the body weight given by most authors as the average. In group 1 (strangulation by encirclement), in which there was only partial arterial occlusion and the loop was resected before the animal died, the loss in the blood volume varied from 20 to 50 per cent, with an average of 35 per cent for the entire group.

In groups 2 and 3, in which there was complete arterial occlusion, there was no increase in the weight of the strangulated loop over the

normal value. Consequently no values for increase in the weight of the intestine are listed for these two groups. In the instances in which the loop was ruptured, the peritoneal cavity contained large amounts of peritoneal fluid of high total protein content, the values ranging from that of the animal's own blood plasma up to twice that amount. In a few instances in which the loop was not ruptured the peritoneal fluid had a total protein content less than that of the blood plasma. The amount of fluid noted in the peritoneum was calculated to be 22 per cent of the total blood volume in the second group and 20 per cent in the third group.

In group 4 (venous ligation), symptoms of shock resulted relatively early and were soon followed by death. Within two hours, the animals began to show a definite increase in the respiratory and cardiac rates, and

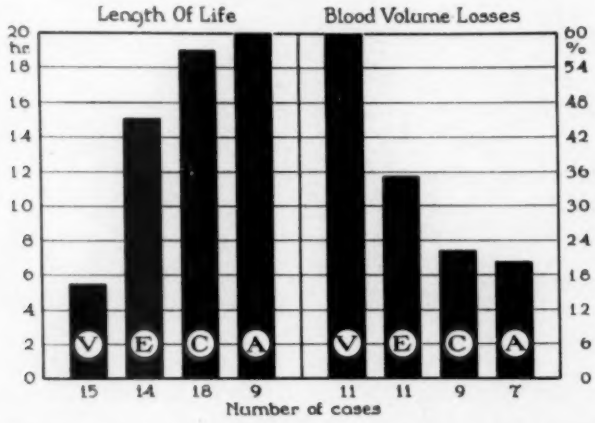

Fig. 5.—The correlation between loss of blood volume and length of survival in dogs with the various types of strangulation obstruction; V = venous; E = encirclement; C = complete; A = arterial.

the hemoglobin content had begun to fall, as had the blood pressure. In a few animals in which the abdomen was opened after this interval, the strangulated intestine was seen to be of a dark mahogany color, lusterless and distended, as previously reported. All these conditions were minimal shortly after the strangulation was produced and increased during its course, reaching a maximum at the time of death. Deaths in group 4 occurred after a loss of from 34 to 66 per cent of the total blood volume. The average for the group was 55 per cent, and the average length of life was five and one-half hours. Losses in the blood volume were calculated as in group 1. A summary of the data will be found in table 8. The rapid fall in blood pressure, early shock and death in group 4 (venous obstruction) were associated with a loss of

blood sufficient in itself to produce these results. The same is true of group 1 (obstruction by encirclement). The results obtained in groups 1 and 4 therefore confirm the work of Blalock,[5] who contended that the fall in blood pressure in shock is due to significant losses in the blood volume. In groups 2 and 3, in which the arteries were ligated, some factor other than loss of whole blood apparently enters in to play the major role in the causation of death.

INTERPRETATION OF RESULTS

The preceding experiments illustrate that strangulation obstruction, like intestinal obstruction in general, is not a distinct disease. The train

TABLE 8.—*Losses in the Blood Volume in Thirty-Eight Dogs With the Various Types of Strangulation Obstruction*

	Weight of Dog, Kg.	Length of Intestine, Ft.	Time of Strangulation, Hr.	Peritoneal Fluid, Cc.	Weight of Intestine, Gm.	Normal Weight of Intestine, Gm.	Percentage Increase in Weight of Intestine	Total Loss of Blood, Gm.	Percentage of Loss in Blood Volume
Group 1									
Minimum	8	3.0	2.6	30	240	76	133	180	20
Maximum	21	3.0	5.6	140	675	150	320	635	50
Average	15	3.2	4.0	82	460	103	235	425	35
Group 2									
Minimum	5	1.0	15.0	40	...	...	...	...	8
Maximum	14	5.0	32.0	325	...	...	...	...	48
Average	9	2.5	20.0	142	...	...	...	...	22
Group 3									
Minimum	8	1.0	15.0	40	...	...	...	...	14
Maximum	20	4.0	24.0	350	...	...	...	...	50
Average	15	2.5	20.0	250	...	...	...	...	20
Group 4									
Minimum	8	3.0	2.5	30	260	90	150	260	34
Maximum	22	5.5	12.0	300	1,030	170	490	1,015	66
Average	15	4.3	5.5	96	680	135	283	643	55

of symptoms, the development of shock and finally the ensuing death depend on at least three factors:

1. Loss of blood into the wall of the bowel, the lumen and the peritoneal cavity.

2. Transudation of plasma into the general peritoneal cavity.

3. Absorption of bacterial "toxins."

The course taken in any strangulation will depend on the relative degree of venous or arterial occlusion. If arterial occlusion predominates, the resulting pathologic picture will be an anemic (in the case of an absolute vascular occlusion, which probably occurs only in massive arterial thromboses) or hemorrhagic necrosis without marked distention of the lumen or the wall of the bowel. In these instances there will be an outpouring of plasma from the surrounding peritoneal surfaces in an attempt to rid the peritoneum of the gangrenous bowel.

Death will occur relatively late and will be secondary to a loss of plasma into the peritoneal cavity and an absorption of bacterial toxins therefrom. If, on the other hand, the venous occlusion predominates in the face of normally patent, pumping arteries, or even partially patent arteries, the result will be a loss of whole blood into the wall and lumen of the bowel and a transudation of plasma into the peritoneal cavity, associated with a distention of the strangulated loop, varying from a moderate to a marked degree.

Of clinical interest is the fact that shock and death result early after pure venous obstruction only when the strangulated intestinal loop is adequately long, or when no egress of blood can take place into the lumen of the adjacent normal bowel to relieve the intraluminary pressure of the strangulated loop. In many clinical cases, an egress of blood does occur into the normal adjacent bowel, and the rate at which blood is lost most likely depends on the degree of obstruction of the lumen of the bowel and on the resulting intraluminary pressure.

SUMMARY

The results of 240 experimental strangulation obstructions are herewith presented. They seem to show that a loss of whole blood and plasma is an important contributing factor to the development of shock and even of death. The gross pathologic picture, as well as the microscopic observations, indicates a loss of whole blood into the wall and lumen of the strangulated loop of the bowel.

The type of strangulation produced is found to be dependent on the relative degree of venous or arterial occlusion. When venous occlusion predominates the loss of whole blood is the chief factor, and when arterial occlusion predominates the loss of plasma is important. Tables are presented which show that there is a marked increase in the weight of the strangulated bowel over the normal weight. This increase in weight has been found to be due to the accumulation of blood in the wall and lumen of the strangulated loop of the bowel. Determinations reveal that the material within the loop has a high hemoglobin content and that the free peritoneal fluid is similar in total protein content to the animal's own blood plasma.

The loss of blood in strangulation obstructions has been calculated and found adequate, with the venous types of occlusion at least, to account for the shock and death which occur. Experiments presented tend to show that the blood pressure falls rapidly and the hemoglobin content drops rather uniformly. Experiments are also presented which show that the fall in blood pressure depends on the length of intestine involved and on the predominate type of strangulation produced.

Experiments are presented to show that "toxic products" are not present in the peritoneal fluid except terminally when the loops are

gangrenous or ruptured. Histamine, strychnine and tetanus toxin have been introduced into strangulated loops in order to test the absorption of these products. Practically all the tests failed to reveal any evidence of the absorption of these products except terminally, when the loops were gangrenous or ruptured. Experiments are also presented which tend to show that the intestinal wall per se is not toxic.

The only evidence of toxic absorption is found late in the course of any strangulation obstruction and is apparently due to the presence of innumerable bacteria in the peritoneal cavity. The effect of the bacteria is apparently quantitative, as gross perforation of the intestine is essential to a lethal issue when the peritoneal fluid is tested by introducing it into the peritoneal cavity of a normal animal.

CONCLUSIONS

1. There is little or no evidence for the direct absorption of "toxic products" from strangulated loops of intestine.

2. There is little evidence for the transperitoneal absorption of "toxic products," except late in the course of strangulation obstruction, when the wall of the bowel is no longer viable.

3. Whole blood and plasma are apparently lost from the general circulation in quantities sufficient in themselves to account for the symptoms of shock and death which occur in most cases of strangulation obstruction.

OSTEOGENIC SARCOMA

A REPORT OF TWO UNUSUAL CASES

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I have been fortunate in observing two interesting cases of osteogenic sarcoma in the past five years, and I submit the records in detail:

REPORT OF CASES

CASE 1.—J. S., a girl aged 13, was first seen in April 1932. The mother stated that several months previously her daughter had fallen, striking her right knee. Sometime later the child began to limp and to complain of pain in the leg. A swelling appeared above and to the outer side of the knee, and the patient was taken to the General Memorial Hospital where she was examined. A diagnosis of osteogenic sarcoma was made, and the family was advised that an amputation was imperative. The parents refused to consent, and a short while later the child was brought to my office.

After having some roentgenograms taken, I made a diagnosis of osteogenic sarcoma and advised amputation (fig. 1). A second refusal led me to plead for local excision, and this was granted. Part of the growth was removed at the Columbus Hospital Extension on May 23, 1932, and the pathologic report of Dr. A. Sala stated that the growth was a rapidly growing osteogenic sarcoma.

Still the family refused consent for an amputation. My only recourse, in the hope of retarding the growth, was to give a series of injections of Coley's toxin. Injections were given locally and in the buttocks for nearly a month, but were of no avail. The pain and limp increased, and the growth soon reappeared locally.

A roentgenogram at this time showed the growth to be as large as ever (fig. 2). Finally the parents consented to an amputation, which was done through the upper third of the thigh on July 17, 1932, at Bellevue Hospital.

The pathologic report, made by Dr. McWhorter, was as follows: "The specimen consists of the lower end of the femur, which has been split longitudinally (fig. 3). On the popliteal surface, just proximal to the epiphysial line, is a fusiform growth of dense consistency. This growth involves the medullary cavity and appears to be growing outward. Microscopic examination shows a densely infiltrating mass of undifferentiated connective tissue cells of short spindle type. Scattered throughout the sections are many giant cells of tumor type" (fig. 4).

Since the operation the patient has slowly recovered, but she has been kept under observation. She has not gained much weight, but periodic roentgenograms of her chest and long bones show no metastases. During the past year she has had

Read before the New York Surgical Society, Oct. 14, 1936.

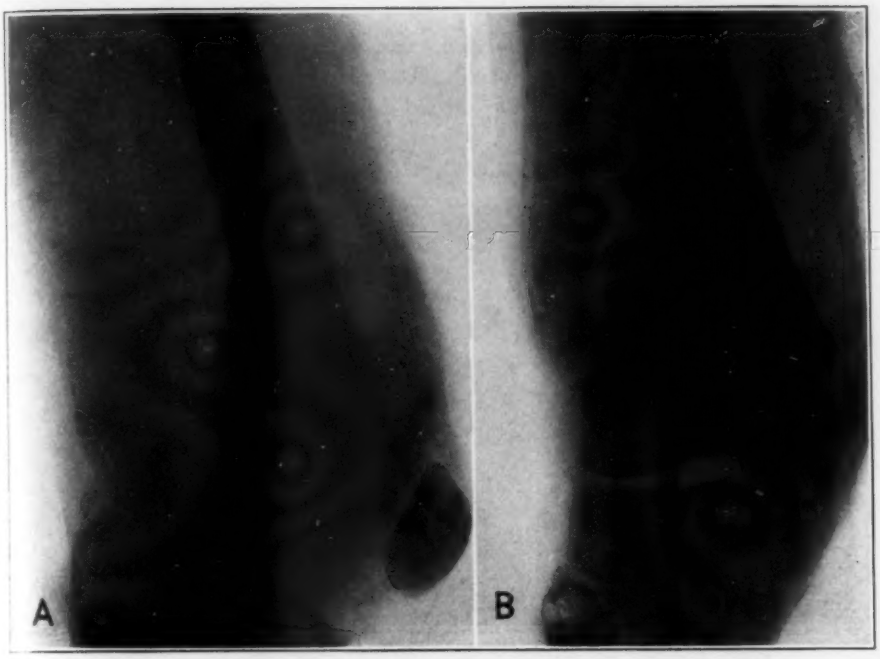

Fig. 1 (case 1).—*A*, lateral view of the lower end of the femur, showing growth that involves the periosteum. *B*, antero-posterior view of the femur, showing characteristic sun ray bony spicules.

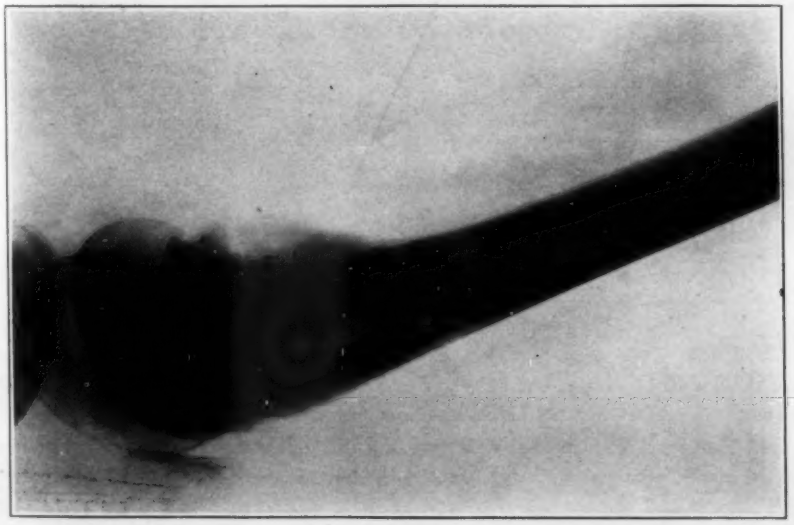

Fig. 2 (case 1).—Lateral view of the femur, showing rapid recurrence following curetting.

trouble with her stump. It has become quite swollen, and she has not been able to wear an artificial limb. A plastic revision was done in April 1936, and a fairly large slice of skin and fat was excised. No evidence of any soft local tumor recurrence was found.

Fig. 3 (case 1).—Cross-section of the specimen.

Case 2.—E. D., a boy, a patient of Dr. D. Zetena, was admitted to the Columbus Hospital Extension Nov. 26, 1934, and was discharged Jan. 27, 1935.

The history given was that while passing through a door of a train on Sept. 30, 1934, the child was pushed by the crowd so that his right foot was caught between the platform and the train and he fell onto the platform, striking his right thigh. From that day on he complained continuously of pain, and his mother noticed a swelling on the right thigh, which was soft, was not red and did not fluctuate.

Fig. 4 (case 1).—Photomicrograph of osteogenic sarcoma of the thigh.

Physical examination revealed nothing abnormal except the painful swelling in the upper part of the right thigh. There had been no previous illnesses except pneumonia at the age of 8 months and otitis media at 1 year.

The blood count on admission showed 13,000 white cells, with 52 per cent lymphocytes and 37 per cent neutrophils. The urine was essentially normal.

Roentgen examination of the right femur showed slight irregularity of the periosteal shadow along the anterior and lateral borders of the upper third of the shaft of the femur, the changes beginning slightly below the greater trochanter and extending downward about 3 inches (9.5 cm.). The periosteum was slightly elevated in the upper and lower portions. There were fine radiating lines extending at a right angle from the cortex into the soft tissue. There was no definite

Fig. 5 (case 2).—Early osteogenic sarcoma with sun ray bony growth into the soft parts.

involvement of the cortex or medulla. The changes had the appearance of a periosteal osteogenic sarcoma (fig. 5).

On Dec. 1, 1934, the patient was ready for disarticulation at the hip; so a preliminary histologic examination of a portion of the periosteum was made, and a frozen section showed a sarcoma. Thereupon disarticulation was carried out.

Two curved incisions were made starting about the center of Poupart's ligament, one going outward and the other inward, both meeting behind near the middle of the gluteal fold. The femoral vessels were exposed and doubly ligated with chromic catgut. The adductor and abductor muscles and hamstrings were severed close to their origin. An injection of 90 per cent alcohol was made into the sciatic nerve, as located, and the nerve was cut high up. The capsule of the joint was incised, the ligamentum teres clamped and divided and the head of

the femur disarticulated. The joint cavity was then thoroughly curetted. All excess muscle was excised, and the rest was approximated by loose interrupted catgut sutures. A drain was introduced down to the acetabulum, and the skin flaps were brought together by interrupted silk sutures.

The patient had moderate fever and was given three transfusions, one of which was necessitated by a fall soon after he began to walk. This fall resulted in a

Fig. 6 (case 2).—Cross-section of the femur, showing osteogenic growth.

slight hemorrhage from the stump. Fifty-seven days after operation the patient was discharged with the stump well healed.

The pathologic report, made by Dr. A. Sala, was as follows: "The upper third of the femur is the seat of a periosteal tumefaction growing outward into the soft tissues (fig. 6). Spicules of bone are palpable, projecting from the periosteum. The upper half of the femur is yellowish in whorls, suggesting neurogenic sarcoma; the lower half is soft and fleshy and grayish. The microscopic diagnosis is osteogenic sarcoma arising from the periosteum."

COMMENT

The first case is reported because of the extraordinary fact that after a rapid local recurrence of the growth following the initial operation, and after repeated injections of Coley's serum which did not help the condition, an amputation was done. At the present time, after an interval of five years, there has been no reappearance of the tumor in the stump or in any other part of the body.

The second case is of interest in view of the fairly definite history of trauma, the uncommon location of the growth and the fact that the child could stand the shock of a radical disarticulation of the hip. This patient has gone over two years without recurrence or metastases.

DUGAS ON THE CURABILITY OF INFLAMMATION

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Dr. Louis Alexander Dugas (1806-1884) held the Chair of Surgery at the Medical College of Augusta (the present University of Georgia School of Medicine) from 1855 to 1882, was dean for a period of twenty years and was editor of the *Southern Medical and Surgical Journal* from 1851 to 1858. Eponymously he is remembered for his famous sign indicative of dislocation of the shoulder joint. During his professional career he published approximately one hundred articles, which are a credit to his surgical versatility and to the wide range of his medical knowledge. The paper which follows is one of several Dugas manuscripts which have recently come to light.

REMARKS ON THE CURABILITY OF INFLAMMATION

BY L. A. DUGAS, M.D.

(Read before the Augusta Library and Medical Society, Sept. 3, 1875.)

What is inflammation?—If we look to Erichsen for a reply we find that "the study of the inflammatory process is one of the most complex and difficult on which the surgeon can enter; but the labor required to master its details is well bestowed, inasmuch as an acquaintance with its nature, symptoms and progress, gives an insight into a greater part of the Science of Surgery."

He then adds: "Yet as the discussion of this subject belongs rather to the domain of General Pathology than to that of Practical Surgery, it cannot consistently be entered upon here" (p. 87-Am. Ed.-1869).

Turn to Gross (p. 49, 1872) and you find that "inflammation may be defined to be a perverted action of the capillary vessels of a part, attended with discoloration, pain, heat, swelling and disordered function, with a tendency to effusion, deposits or new products. In addition to these changes, there is also an altered condition of the blood and nervous fluid, as an important element of the morbid process. In what inflammation essentially consists, it is as impossible to determine as it is to explain the intimate character of attraction, repulsion, gravitation, or cohesion."

Hear Ashurst: "Authors, though differing as to the proper explanation to be given of many of the phenomena of inflammation, are, I think, generally agreed that those phenomena are mere modifications of the phenomena of natural textural life." (p. 34).

It is not necessary to multiply references to authorities, for this would be simply to make confusion worse confounded. No attempt at a mere definition of inflammation can be successful. We must first recognize and study the funda-

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mental physiological functions of the body, that is to say, Innervation, Capillary Circulation, Nutrition and Secretion, and we shall find that inflammation always involves a deviation from the normal condition of every one of these functions.

While there may be lesions of any one of these functions, or of several of them, without inflammation, this *always* exists when the whole are affected. And yet inflammations are not always alike; they differ infinitely according to their cause and to the structure invaded.

Inflammatory affections, so called, are therefore as numerous and as various as the diseases classed under this head. No two are alike in their manifestations, nor in their history. They differ in causation, in symptoms, in duration, in terminations and in curability. They are therefore strictly speaking, entities; and should be more generally admitted to be so. If this general assent could prevail we might dispense with the word inflammation, and allow each entity or disease to be judged and treated according to its own or individual peculiarities. But the word has been so long in use, and is so intimately interwoven with our facts as well as theories, that we may not expect to see it set aside in our generation, nor probably in several more of them. With this explanation, I will continue its use as a matter of convenience whenever it answers the purpose.

Is inflammation curable?–This may strike some as a ridiculous question, for it is in striking contrast with the dogmatic declarations of every period of the history of medicine. To intimate any doubt as to the efficacy of the modes of treatment advocated by leading men from Hippocrates to Broussais, would seem to be as preposterous in Medicine as heresy in theology. And yet the extravagancies of Broussais aroused a spirit of inquiry so potent that he lived to see his favorite dogmas, first doubted, and then almost unanimously discarded by the profession. His hobby that by depletion all inflammations could be readily "removed," has now become obsolete, and every one knows that you may draw blood "ad deliquium animi" without curing a simple pimple on the face, and that the same inefficiency attaches to any other form of antiphlogistic treatment. I wish it to be borne in mind that I use the term *cured* in its strict sense; that is to say, that the inflammatory process was found to pursue the even tenor of its way in despite of any known form of treatment.

As it was my good fortune to be at the seat of war whilst it progressed most furiously between the friends and adversaries of the Broussaisian School, I had abundant opportunities to verify the claims of the respective parties; and became satisfied that the victory was with the opposition, and that inflammation could not be *cured* by antiphlogistics, nor by any other plan of treatment then known. Whenever the inflammation was on the surface, so as to be seen in its various stages, it was never arrested by treatment; but ran its peculiar regular course to resolution or to some other of its accustomed terminations. Such being the case with regard to inflammations subject to ocular inspection, we could not reasonably suppose different results in the progress of inflammations affecting internal organs.

Such are the doctrines I continued to teach until about ten years ago; and the object of this paper is to lay before you how my views have undergone a change.

As far back as the introduction of Quinine in the treatment of our malarial fevers, which were then considered inflammatory, and treated as such, I was forcibly impressed by the prompt and certain arrest of the disease by this wonderful agent. Could it be that quinine arrested inflammation?–or had we been in error with regard to the pathology of our fevers?–The organs apparently implicated were out of sight, and we might have mistaken hyperaemia for inflammation. I became convinced that such was the fact, and the efficacy of Quinine

was to be found in its effect on the nervous system, the blood, and the capillary circulation in general; thus bringing about an equalization of circulation and relief of *congestion*—not *inflammation*. There is no lack of evidence that by the timely use of quinine we may *prevent* the development of inflammation in cases in which this would probably have occurred without it. We have also strong reasons for believing that quinine may modify the progress of inflammatory action after its occurrence. But I have yet to see a case in which inflammation once set up, has been "jugulated" by quinine. It is true that quinine exerts a most beneficial effect upon Pneumonia and Dysentery as they prevail in this section of the country, and that it is really our sheet-anchor in these affections. But this is so because our forms of pneumonia and dysentery, especially when epidemic, are almost invariably mixed with malarial or paroxysmal fever, which readily yields to quinine, and leaves the inflammatory complication to subside gradually under judicious management.

About ten years ago I found that by applying Tincture of Iodine to a furuncle the progress of inflammation was arrested and it terminated by resolution. Repeated trials were attended with similar results. If applied even so late as the formation of the core, the pain would cease, the swelling would subside, and the core would come away in due time. You know that furuncles are of two kinds, the one simple and the other preceded by a vesicle, and therefore called the vesicular. From the resemblance of the latter form to carbuncles, I formerly designated them as *carbuncular furuncles*, but now prefer to call them *vesicular furuncles*, because this indicates their anatomical peculiarity. Moreover, carbuncles never begin with a vesicle. Vesicular furuncles are much more slow, painful and extensive than simple furuncles, and have a predilection for the back of the neck, where they are frequently mistaken for carbuncles. If you will closely examine them in the initial stage, you will scarcely ever fail to detect a vesicle about the size of a pin-head, attended with itching, and subsequently with tumefaction and pain. The vesicle is soon rubbed away by attempts to relieve the itching. The vesicle does not reappear, but the inflammation extends through the skin into the subjacent tissue, terminating in suppuration and sloughing.

Now if you apply the Tincture of Iodine to such cases in any stage you may safely expect to put a stop to any further inflammatory action. Both pain and tumefaction will readily subside and the trying use of the knife be obviated.

I have never had an opportunity to test the efficacy of this treatment in genuine carbuncle; but am strongly disposed to think it might be equally beneficial as it is in furuncles.

But a much more useful application of this treatment is to be found in Erysipelas, especially when consequent upon traumatic causes. The relief in such cases is sometimes so prompt as to partake of the marvelous. The last case I treated was that of a man who had sustained an injury to one of his fingers and came to me with the whole hand greatly swollen, red and painful, with rapidly spreading erysipelas. I immediately gave the entire red surface a thorough painting with Tincture of Iodine, and advised its repetition in 6 and 12 hours. He visited me after the third painting, and I found the tumefaction much reduced, no pain, no tendency to spread, and every indication of the rapid subsidence of the disease. He was advised to use the application only morning and night, and was well in a few days. This plan of treatment may be regarded as specific, when resorted to sufficiently early and in the proper way.

For furuncles and erysipelas I usually direct the painting to be effectually done morning and night, in bad cases three times a day, and gradually discontinued as the disease disappears. In the sub-cuticular and sub-cutaneous forms of whitlow

I have found Tincture of Iodine sometimes beneficial, but not so in the thecal and periosteal varieties. For buboes, whether syphilitic or otherwise, I have no reason to think it prevents or lessens the tendency to suppuration; indeed I am rather disposed to think I have seen suppuration oftener when it was used than when I resorted to other expedients. This may appear singular when we remember how valuable an agent the Tincture is in dispensing some of the chronic enlargements of lymphatic glands.

We have endeavored to demonstrate; first, that no *definition* of the word inflammation hitherto proposed is satisfactory.

Secondly, that the so-called inflammation is a radical perturbation of the fundamental physiological functions, which varies according to its cause and the tissues involved;

Thirdly, that inflammations should be regarded and treated as entities, or distinct diseases;

Fourthly, that when I commenced my professional studies it was generally conceded that inflammatory affections were curable;

Fifthly, that the ultraisms of Broussais instigated a spirit of inquiry which resulted in the conviction that no treatment then known could be said strictly to cure or to arrest the regular progress of inflammatory action;

Sixthly, that this conviction became, with me, somewhat shaken by the introduction of quinine;

And finally, that it is now demonstrated beyond doubt that some of the forms of inflammation may be effectually arrested by the application of tincture of iodine to the affected locality.

It has been wisely stated that there is nothing new under the sun, and I shall therefore not lay claims to originality which might be controverted by the more erudite; but I must say that my first use of Tincture of Iodine, as above narrated, was not instigated by knowledge derived from others—If others had done the same before I was not aware of the fact.

I have ventured to place this paper at your disposal because I regard the discovery—by whomsoever made—of the fact, that some forms of inflammation are undoubtedly curable, as one of the utmost importance. It not only corrects one of the convictions of the learned, but must lead to farther discoveries in the same direction. As one step in advance is only the precursor of others, let us redouble our exertions, in the hope that some one among us may have the honor of contributing the next fact.

PARTIAL CHOLECYSTECTOMY

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The treatment for acute suppurative or gangrenous cholecystitis has recently been given renewed consideration. The controversial question has again been raised of immediate or early operation as against delay in operation, because the mortality following early operation has seemed to be lower than that following routine operative delay (Heuer). The operation of choice for acute or chronic cholecystic disease is removal of the gallbladder. One may expect a high mortality if cholecystectomy is invariably used on patients who are extremely ill or who are bad risks, and for such patients cholecystostomy, which is merely a palliative procedure in from 25 to 30 per cent of cases, must be resorted to. The likelihood that cholecystectomy must be performed subsequent to cholecystostomy because of recurrent symptoms or a persistent biliary fistula makes primary cholecystectomy particularly desirable. In cases of suppuration, cholecystectomy from above downward often proves to be easier. But when there is massive inflammation and induration about the cystic and common ducts, any type of complete cholecystectomy may be not only technically difficult but attended with difficulty in controlling hemorrhage and accompanied by an unduly high mortality. It is particularly for this type of severe suppurative or gangrenous gallbladder that the procedure of partial cholecystectomy has been found to be exceedingly valuable.

OPERATIVE PROCEDURE

The operative technic follows:

The gallbladder is exposed by an incision through the upper right rectus muscle or one parallel to the costal margin. The area involved is isolated by gauze packs, and the adhesions to the gallbladder are freed. The gallbladder is aspirated of its fluid contents, as shown in *A* of the accompanying illustration. The fundus is incised, the stones are removed and the gallbladder is dried with gauze and swabbed with tincture of iodine; it is then split down with scissors to within from 1 to 2 cm. of the cystic duct, as in *B;* this often facilitates the removal of a stone impacted at the junction of the ampulla and the cystic duct which cannot be evacuated otherwise. The gallbladder is partially removed by trimming off the redundant part of each half down to the border of the fossa of the liver, that portion which is attached to the liver being left, as in *C*. The bleeding from these cut edges is controlled by

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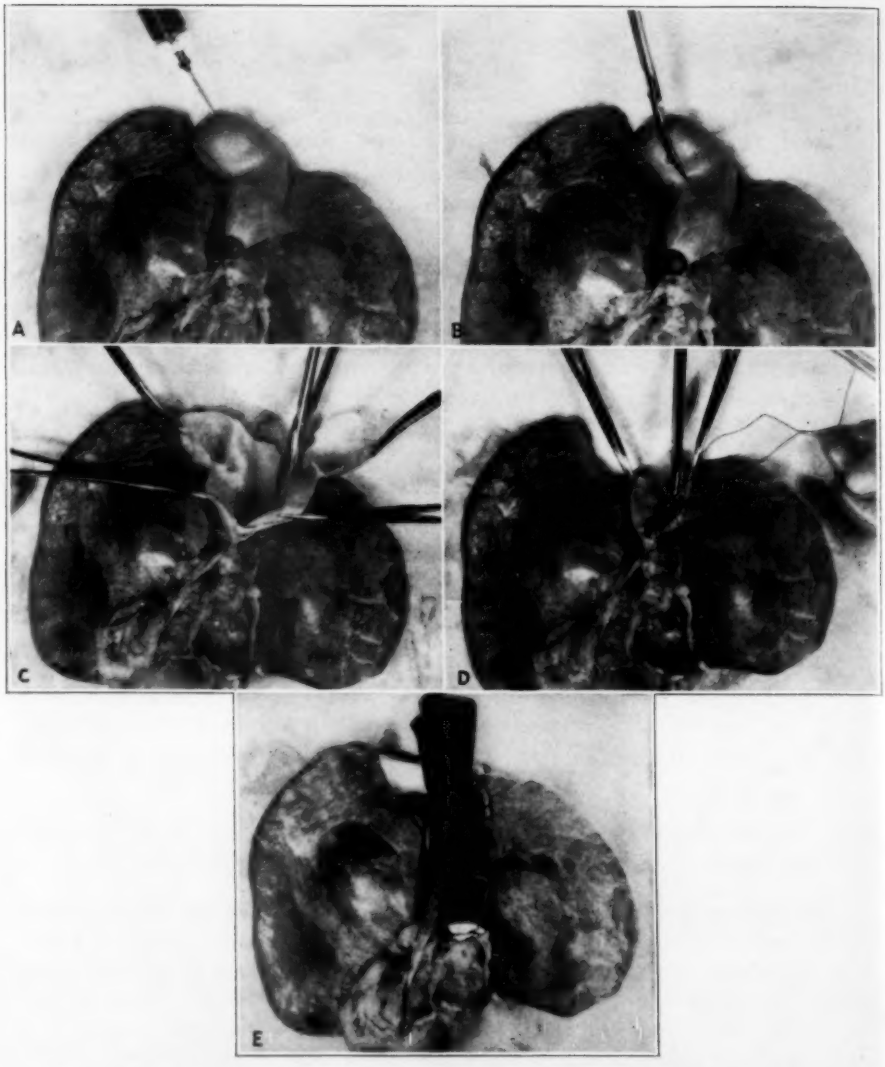

Steps in the operative technic. The gallbladder is first aspirated, as in *A*, and the fundus is incised, as in *B*. After the fundus is incised, the stones are evacuated, the gallbladder is dried out and swabbed with tincture of iodine and is split down to the cystic duct. After the removal of any stone at the junction of the ampulla and the cystic duct, the redundant portion of each half is trimmed off with scissors, leaving only that portion of the gallbladder attached to the liver, as in *C*. The cut margins of this remnant of the gallbladder are sutured with a lock stitch, as in *D*. Cigaret drains are placed about the cystic duct and against the remnant of the gallbladder to hold it open, as in *E*.

ligature or lock stitch up each side, as in *D*. Two or three cigaret drains are placed about the cystic duct and brought out against the remnant of the gallbladder to hold it open, as in *E*. A tube may be placed at the opening of the cystic duct, which is not ligated or tied off in any way. The greater omentum or the thickened plastic gastrocolic omentum is placed against the drains to separate them from the nearby viscera. The drains are brought out either through the operative wound or through a lateral stab wound directly over the remnant of the gallbladder, and the wound is closed.

I have used pure phenol or actual cautery in a few cases instead of tincture of iodine to destroy the mucous membrane of the remaining bit of gallbladder. But having found that there was no evidence of reformation of the gallbladder in the early cases in which iodine was used, I have recently uniformly applied it to avoid the possibility of prolonging the convalescence by the development of an eschar or necrosis, as in one case in which cautery was employed.

INDICATIONS FOR THE OPERATION AND MORTALITY

In 1929 I described this operation and reported the end-results in seven cases.[1] I have now employed it in forty-eight cases over a period of fourteen years. In forty-four cases it was used because of an acute suppurating or gangrenous gallbladder with induration about the cystic and common ducts. In four, it was used because of extremely difficult exposure of a small contracted gallbladder which was densely adherent to the liver.

There has been one death, a mortality of 2.08 per cent. This fatality occurred in a woman 65 years of age with advanced myocardial disease and a large gangrenous gallbladder. She had recovered, apparently, and was about to be discharged from the hospital on the sixteenth postoperative day, when a large pulmonary embolus developed.

FOLLOW-UP STATISTICS

A careful personal follow-up examination has been obtained on all but five patients, i. e., on forty-two.

Relief from Symptoms.—Thirty-four patients (81 per cent) have remained well and free from any complaint referable to the gallbladder, the longest for twelve years. Five (11.9 per cent) require a slight dietary restriction, a diet low in fat, because of occasional bloating and belching after meals. No biliary fistulas have occurred, and no operation has been performed for recurrent disease of the gallbladder. Frequently, after the third or the fourth postoperative day there was a free discharge of bile, but the sinus healed readily after the drains were removed.

Subsequent Involvement of the Common Duct.—Three patients (7 per cent) have required operation for stones in the common duct six, eight and nine years, respectively, after partial cholecystectomy.

1. Estes, W. L., Jr.: Partial Cholecystectomy, Arch. Surg. **23**:119 (July) 1931.

Also, two of the five patients requiring dietary restrictions have had transitory symptoms of jaundice, epigastric pain and vomiting at long intervals. One of these has remained free from symptoms for eight years and now has diabetes. Another patient had ten stones removed from the common duct coincident with partial cholecystectomy, but as a rule simultaneous exploration or operation on the common duct is rarely advisable when extensive induration and inflammation are present about the duct because of the added operative risk and excess trauma due to the technical difficulties of exposure and control of hemorrhage and infection. Ordinarily, if the involvement of the common duct is definite at the time of partial cholecystectomy, a two stage procedure had best be planned.

3. *Fate of Remnant of Gallbladder Allowed to Remain.*—I have had the opportunity to reoperate on three patients. Two required hysterectomy, and the area of the gallbladder was simply palpated. There were dense adhesions to the fossa of the gallbladder, but no evidence of any thickening to suggest reformation of the gallbladder. The third patient had to undergo choledochostomy for the removal of 5 stones eight years after the partial cholecystectomy. There were adhesions of the gastrocolic omentum and thickening of the capsule of the liver over the fossa of the gallbladder but no vestige of anything that even resembled the gallbladder or a remnant of the gallbladder.

Postoperative Hernia.—As extensive drainage is necessary in partial cholecystectomy—at least three drains are usually required—postoperative hernia might readily be expected, except in those cases in which the drains are brought out through a lateral stab incision. In my series of cases, an opening in the fascia at the point of drainage could be palpated in most of the scars, but in only six of the cases (14.3 per cent) was there definite hernia—that in an obese man of 76 was large and gave symptoms of obstruction, but he has consistently refused operative relief.

Miscellaneous.—In three cases diabetes developed from three to eight years after operation. In one a keloid occurred in the scar. Six patients have died of disease unrelated to the biliary passages or liver from one and one-half to twelve years subsequent to operation.

REVIEW OF LITERATURE

Partial cholecystectomy is not a new procedure. In 1899 and again in 1900 W. J. Mayo[2] reported cases, particularly of obstruction of the cystic duct, in which the removal of stones and the mucous membrane

2. Mayo, W. J., and Mayo, C. H.: A Collection of Papers, Published Previous to 1909, Philadelphia, W. B. Saunders Company, 1912, vol. 1, pp. 348 and 355.

of the gallbladder was done instead of complete cholecystectomy. Partial cholecystectomy was described in 1920 by Bengolea.[3] In 1921 Martin[4] cited five cases of acute cholecystitis in which he removed the stones, split the gallbladder and held it wide open with drains.

In 1923 de Martel[5] reported that in two of a series of fifty-two operations on the gallbladder in which it was difficult to separate the ampulla and the cystic duct from adjacent structures he split the gallbladder from the fundus to the ampulla, divided the cystic duct and cut away the free part of the gallbladder, swabbed the remaining portion with tincture of iodine and drained it. Both patients recovered.

In 1924 Zabala and Bengolea[6] cited four cases in which partial cholecystectomy was performed, with recovery; in one there was a mass of adhesions about the gallbladder; in another a calculus was impacted in the cystic duct, which was firmly indurated and adherent to the common duct, and in a third a piece of the gallbladder was left near the cystic duct because of the friability of the tissue. They expressed the belief that the operation is indicated in cases of severe suppuration in which resection would be fatal. The gallbladder is split, and the stones are removed, after which the gallbladder is resected, the portion attached to the liver being left; the mucosa of this remnant is curetted, and tincture of iodine is applied.

Pauchet[7] decided that partial cholecystectomy is indicated (1) in the feeble or icteric patient with stones in the common duct and gallbladder; (2) in the patient with an atrophic gallbladder and stones; (3) in the patient with a subacute or acutely inflamed gallbladder adherent to the adjacent organs, and (4) in the patient with suppurative cholecystitis with fever. He expressed the belief that this operation avoids cholecystectomy at a time when complete cholecystectomy would give a mortality of 50 per cent. He followed Zabala and Bengolea's technic, except that the remaining mucosa is swabbed with ether as well as with tincture of iodine, and when there are strong adhesions to the nearby intestines, the gallbladder is freed from the liver and the portion adherent to the intestine is allowed to remain. The operation has been performed only in carefully selected cases in which routine cholecystectomy seemed to be indicated but would be a dangerous procedure because of technical difficulties or the amount of infection present.

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3. Bengolea, A. J.: Bol. y trab. de la Soc. de cir. de Buenos Aires, 1920, p. 285.

4. Martin, E. D.: New Orleans M. & S. J. **74**:204, 1921.

5. de Martel, T.: Bull. et mém. Soc. de chir. de Paris **49**:306, 1923.

6. Zabala, A., and Bengolea, A. J.: Lyon chir. **21**:281 (May 7) 1924.

7. Pauchet, V.: Gaz. d. hôp. **87**:1077, 1924.

In 1927 Gatch [8] and also Zimmerman [9] reported a similar technic.

In performing partial cholecystectomy for gangrenous cholecystitis, Haggard [10] (1930) destroys the mucosa of the remaining portion of the gallbladder either with phenol or with actual cautery, inserts a tube into the cystic duct for drainage and sutures the cut edges of the gallbladder over it.

McKenty [11] (1933), in reviewing the treatment for acute cholecystitis, cited thirty-three cases in which partial cholecystectomy was performed, with one death. After aspirating the contents of the gallbladder, he opens it, removes the stones and swabs the gallbladder with iodine, leaving an iodine pack in "the sac," and exposes and ties the cystic duct and artery. "The sac is then excised either from above downward or from below upward, leaving the portion attached to the liver undisturbed. From this portion the softened mucosa is easily removed by rubbing with gauze preferably saturated with iodine."

Ritchie [12] (1937), after splitting the gallbladder and excising the "wings," removes the mucous membrane of the portion allowed to remain, sutures a tube into the stump of the ampulla and cystic duct and closes the denuded fragment of the gallbladder by suture. He reported sixteen cases in which this procedure had been used, with no mortality.

SUMMARY

It must be understood that this operation would seem to be particularly applicable in cases of acute suppurative or gangrenous cholecystitis or empyema of the gallbladder, especially when there is induration about the cystic and common ducts, and occasionally for the small, densely adherent, thickened, atrophic gallbladder which cannot be easily separated from the bed of the liver. In no way should partial cholecystectomy be considered to supplant complete cholecystectomy when complete removal can be safely accomplished. The operator who is exceptionally skilful in the use of cholecystectomy from above downward may find less use for partial cholecystectomy than one whose proficiency has been directed to cholecystectomy from below upward. Furthermore, though cholecystostomy must still be reserved for the patient who is an exceptionally bad risk, fewer cholecystostomies seem indicated when familiarity with partial cholecystectomy has been acquired.

8. Gatch, W. D.: Tr. West. S. A. **37**:345, 1927.

9. Zimmerman: Tr. West. S. A. **37**:341, 1927.

10. Haggard, W. D.: Wisconsin M. J. **29**:683, 1930; Ann. Surg. **105**:790, 1937.

11. McKenty, J.: Canad. M. A. J. **33**:771, 1933.

12. Ritchie, H. P.: Surgery **1**:581, 1937.

However, what would seem particularly to recommend partial cholecystectomy for acute suppurative cholecystitis is its low mortality. Postoperative mortality from this disease has been reported as 13.5 per cent by Miller,[13] 9.3 per cent by Smith,[14] 5.5 per cent by Graham,[15] 4.7 per cent by Judd and Phillips [16] and 3.2 per cent by Heuer.[17] Heuer, in advising the consideration of an early operation in cases of cholecystitis, especially has pointed out that operation before gangrene or perforation has occurred may play an important role in reducing the mortality. But none of my patients was admitted to the hospital within forty-eight hours of the onset of symptoms. They all received careful routine preoperative preparation and were operated on perforce, therefore not in the early stages of the disease. Partial cholecystectomy under these conditions, yielding a mortality of but 2.08 per cent, would seem to have a place in the treatment of advanced gangrenous or suppurative cholecystitis.

CONCLUSIONS

1. Partial cholecystectomy has and should have a very restricted field.

2. It should not be used when complete cholecystectomy can safely be performed.

3. It is particularly indicated: (*a*) in cases of acute gangrenous and suppurative cholecystitis when complete cholecystectomy is desirable but is technically impossible or unsafe, particularly with extensive induration and infiltration about the cystic and common ducts, and (*b*) in an occasional case of a contracted thickened gallbladder which is markedly adherent to the liver.

4. End-results in forty-eight cases would seem to demonstrate: (*a*) that partial cholecystectomy does act as cholecystectomy, i. e., no reformation of the gallbladder or stones occurs subsequently; (*b*) that it would seem to give end-results comparable to those for cholecystectomy in cases of cholelithiasis (81 per cent of patients completely well and 11.9 per cent well with dietary restrictions); (*c*) that postoperative involvement of the common duct may occur occasionally—7 per cent, and (*d*) that when carefully and reasonably employed, the operation is attended by a surprisingly low mortality.

ABSTRACT OF DISCUSSION

DR. WILLIAM D. HAGGARD, Nashville, Tenn.: The indications and contraindications for partial cholecystectomy have been well outlined. It is a modus vivendi remedy, designed to do away with the serious complications that occur after cholecystectomy of the gangrenous gallbladder.

13. Miller, R. H.: Ann. Surg. **92**:644, 1930.
14. Smith, M. K.: Ann. Surg. **98**:766, 1933.
15. Graham, H. F.: Ann. Surg. **93**:1152, 1931.
16. Judd, E. S., and Phillips, J. R.: Ann. Surg. **98**:771, 1933.
17. Heuer, G. J.: West Virginia M. J. **26**:11, 1930; Ann. Surg. **105**:758, 1937.

The large, edematous, water-logged gallbladder renders isolation of the cystic duct difficult. Removal of the gallbladder, even from above, entails considerable risk of injury to the deeper ducts. Of all things that one wishes to avoid, that evil result is the least desirable. Partial cholecystectomy will obviate that danger. In the large gangrenous gallbladder which is so friable one can scarcely handle it without rupture and soiling, aspiration and adequate tamponade are essential.

Excision of the diseased or gangrenous wall of the gallbladder leaves the part that is embedded in the fossa of the liver. Having the best circulation on account of its attachment to the liver, it is rarely gangrenous. One need not be afraid of the little part that is left. The surgeon is so imbued with the idea that he must take out every particle of the gallbladder that he is fearful about leaving any part of it.

After the stone or stones and the liquid contents have been removed, the walls of the gallbladder are cut away. Removal of the remnant adherent to the liver is often a bloody procedure and leaves a large raw bleeding surface that is difficult to suture. Multiple drains are usually required. The little piece of gallbladder adherent to the bed of the liver is the patient's protection.

One can remove the mucosa by rubbing it with gauze. Swabbing with phenol will denude it, and the acid is then neutralized with alcohol. Destruction of the mucosa will do away with secretions, and no sinus will result.

The cut edges of the remnant of gallbladder are sewn over a catheter in the cystic duct. Drainage is adequate. It is a most satisfactory way to get out of a difficult situation and avoid adhesions.

Dr. Donald Guthrie, Sayre, Pa.: I agree that the operation Dr. Estes has described is the correct one to employ in these desperate situations instead of the more radical operation of cholecystectomy.

The surgeon should use his best judgment in the selection of cases of cholecystitis for operation during the acute phase of the disease, for in spite of all that has been said lately about the need of prompt operation to prevent perforation or gangrene, the tendency is toward conservative treatment. Neither gangrene nor perforation of the gallbladder are feared as much in cases of cholecystitis as in cases of acute appendicitis.

My associates and I have practiced in a measure the plan suggested by Dr. Estes. We remove the mucous membrane and leave the serosa behind, for infection cannot recur in a hollow viscus if the mucous membrane is removed or destroyed.

This procedure is called a compromise cholecystectomy. After the contents of the gallbladder are aspirated and the gallbladder is opened, the organ is split from the fundus to the infundibulum, the margins are widely retracted and the thickened mucous membrane is separated from the cut edges with the handle of the scalpel.

After the mucous membrane is well separated from these edges, it is usually easy to complete its removal with finger dissection. Bleeding vessels are ligated, excess serosa is cut away and the edges are sewed together over a small rubber tube.

It is usually impossible to bring the edges of the liver together after complete removal of a large gallbladder, because the thickened posterior layer of the serosa generally comes away with the gallbladder, and it is often necessary to pack the bed of the liver with gauze to control the bleeding. This is dangerous.

Poor exposure, improper mobilization of the infundibulum and hemorrhage from the cystic artery are frequent causes of injury of the common duct, with the development of a postoperative biliary fistula or a stricture of the common duct.

The plan of Dr. Estes deserves more general adoption, for I believe that the surgeon should be less prone to perform cholecystectomy in the presence of fulminating cholecystitis in an extremely ill patient, and the partial operation has proved to be safe. Furthermore, it is curative, as is so well proved by Dr. Estes' reported cases.

DR. MOSES BEHREND, Philadelphia: I have never been compelled to use this technic. I have used it in cases of chronic cholecystitis simply for teaching purposes, but otherwise I always follow the technic of Max Thorek, of Chicago. I believe that much can be gained by electrocoagulation of the exposed surface of the mucous membrane in place of the use of phenol or actual cautery.

I never use drainage in these cases, but sew the falciform ligament over the fulgurated mucous membrane. In fact, I do not use drainage in any case of cholecystectomy, because I believe that the convalescence is better without it. Formerly I employed drains, but in the last fifteen or twenty years I have discontinued their use entirely.

Dr. Estes opened up an enormous subject when he spoke about operating in cases of acute cholecystitis. I do not believe that he meant one should operate in such cases as an emergency measure. I never performed an emergency operation (and I agree with Dr. Guthrie on this point) in a case of acute cholecystitis or acute empyema of the gallbladder. I wait from eight to ten and even twelve days until the symptoms have subsided. I have never been compelled, therefore, to use the technic described by Dr. Estes.

Even in cases in which there is a hard peritoneal covering over the cystic and common ducts, it is necessary only to make an incision with a sharp knife in the line of direction of the ducts and then to separate the peritoneum with long flat scissors. The ducts can be exposed readily, and an excellent cholecystectomy can be performed.

My mortality is from 1 to 2 per cent. I believe that this is due to the fine exposure of the field of operation and to the fact that I never operate in cases of acute cholecystitis or of acute empyema as an emergency measure.

DR. WILLIAM L. ESTES JR., Bethlehem, Pa.: My deep thanks go to Drs. Haggard, Guthrie and Behrend for their illuminating discussions.

To make it perfectly plain in referring to partial cholecystectomy and its indications, I repeat that this procedure is to be used only in a rather restricted field. It should be used in cases of advanced, acute, suppurative or gangrenous cholecystitis.

I do not consider it an emergency operation. And whereas this is a controversial field, my own feeling is that one should distinguish definitely between an emergency operation and early operation for acute cholecystitis. In other words, a patient with acute suppurative cholecystitis should be properly prepared for operation and not rushed to the operating room; but if he is in a satisfactory condition, he may be operated on early. Partial cholecystectomy is one of the procedures that is applicable in the treatment.

FRACTURES OF BOTH BONES OF THE LEG

MANAGEMENT BY USE OF THE DOUBLE STEEL PIN TRACTION IN PLASTER OF PARIS

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AND
E. STEWART TAYLOR, M.D.
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This paper is a summary of the treatment, progress and results of treatment of tibiofibular fractures in 23 consecutive cases managed by the traumatic surgery staff at the Hurley Hospital between Jan. 24, 1935, and Jan. 29, 1937. We have had 7 more cases since the last-named date, but we consider these too recent to be useful in drawing definite conclusions. However, thus far the course has been comparable to that in cases in which we have been able to collect more nearly complete data. In addition, other surgeons on the hospital staff have had 17 cases, which are also not included in this series. Results have been uniformly good as far as can be ascertained.

Enthusiasm for the acceptance of the method of management of tibiofibular fractures by means of the double steel pin came as a result of the work of Dr. R. A. Griswold, of the department of surgery of the Louisville City Hospital and University of Louisville School of Medicine, who described the routine procedure, after-care, results and conclusions in the management of 43 fractures of this nature. The results were considered excellent, and one of us (Curry) decided to adopt the method. While a series of 23 cases may appear rather small, the results obtained seem to justify a report at this time.

In the short period of two years the fundamental principle of traction by steel pins, one through the os calcis and the other through the tibia posterior to the tibial tubercle, has been changed only by the availability at present of specialized apparatus for gaining the approximation of the bone fragments.

In this clinic the indications for the use of the double steel pin method of traction and immobilization in plaster of paris for combined fractures of the tibia and fibula are (1) fracture of both bones of the leg and (2) absence of involvement of any joint.

The treatment used consists, as mentioned, of the insertion of a ⅛ inch (0.3 cm.) steel pin through the upper end of the tibia on a level

From the Department of Traumatic Surgery, Hurley Hospital.

with the anterior tibial tubercle and into the substance of the tibia on a plane approximately 1½ inches (3.7 cm.) posterior to the anterior plane of the tibial tubercle. The ends of the pin protrude conveniently about 1½ inches from the surface of the skin on each side. The second, or distal, pin, somewhat shorter, is placed through the body of the os calcis at a point about 1 inch (2.5 cm.) below the external and internal malleoli and slightly posterior to them. The pins are inserted with the patient under either local or general anesthesia. The leg is then lifted, and the ends of the pins are placed in removable

Fig. 1.—Zimmer apparatus used in gaining anatomic apposition of bone fragments. The apparatus is so constructed that changes in length, rotation and height may all be effected by adjustment. A roentgenogram is taken at this stage, or fluoroscopic control is used. A plaster of paris dressing is applied from the toes to the middle of the thigh before the leg, with pins, is removed from the apparatus.

sockets, which are anchored in slits on the proximal and distal portions of the traction apparatus as designed by Zimmer (fig. 1). The clinical alinement of the leg is obtained by rotation of either the upper or the lower fragments and their pin. Traction is exerted by the mechanical attachment for this purpose until the desired length is obtained and until clinical observation, laterally, anteriorly and posteriorly, determines the correct position and length. This is checked by means of a

portable x-ray apparatus; the roentgenogram is developed immediately and is interpreted before the fracture is fixed in plaster of paris. Fluoroscopic control is also recommended, to determine the proper alinement. Plaster of paris is then applied from the base of the toes to the middle third of the thigh, with the foot held at right angles midway

Fig. 2.—A typical tibiofibular fracture, at the time of the patient's admission to the hospital. It fulfils the indication for management with the double steel pin.

between eversion and inversion and with the knee in slight flexion. Pins are incorporated in the plaster of paris, their protruding ends being covered with corks and likewise incorporated in plaster of paris (fig. 3). A walking iron is then applied.

In compound fractures, a débridement of the wound together with a thorough irrigation is done, and the wound is closed. From this point it is treated like a simple fracture. Tetanus antitoxin and gas bacillus antitoxin are given as a part of the routine in all cases of compound

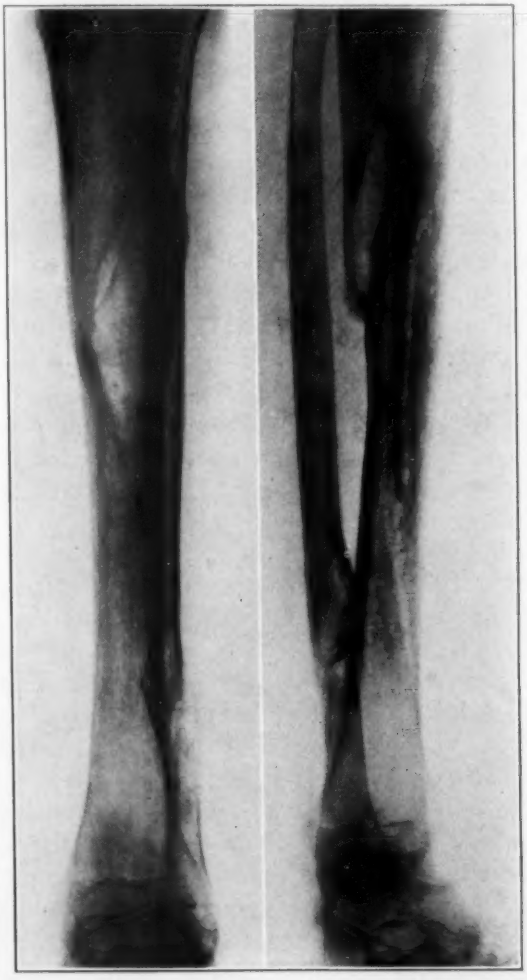

Fig. 3.—The fracture has been reduced by the traction apparatus shown in figure 1. Note that the leg is now in plaster of paris and that the pins remain in place.

fracture. If the fracture is presented late and secondary infection is present, a steel pin is placed through the os calcis for the purpose of traction with weights. When the wound has healed sufficiently the proximal pin is inserted as has been described and a plaster of paris

dressing is applied, incorporating both pins. The patient is kept in bed until his temperature is normal and there are no complications.

As to the etiology of fracture in this series of cases, 10 of the 23 fractures were caused by the patients' having been struck by an automobile; 5 were the result of accidents to persons riding in automobiles; 6 were due to falls, and 2 were the result of farm accidents.

In this series of cases, 10 fractures were compound and the remaining 13 were simple. The roentgenologic diagnosis of these 23 fractures classified 20 as comminuted fractures of the tibia and 3 as spiral fractures of the same bone. In each case the roentgenogram showed an accompanying fracture of the fibula, either at the same level or in the upper third. As to the location of the tibial fractures, 7 occurred at the junction of the upper and the middle third of the shaft, 5 at the junction of the lower and the middle third, 4 in the middle third, 3 in the lower third and 1 in the upper third. One tibia showed fractures at the upper and the middle third, 1 had 2 fractures involving the middle and the lower third, and another had 3 fractures in the upper half of the tibial shaft.

All but 4 of the patients were operated on within a period varying between two and five days after the occurrence of the accident. The operations for these 4 patients were necessarily delayed on account of other major injuries.

One of the outstanding features of this method of treatment is the materially shortened period which elapses before the patient is up and about after the operation (fig. 4). For 19 patients the average time spent in bed after the operation was six days. At the end of this time they were discharged on crutches. The number of days from operation to discharge varied from one to thirteen. The remaining 4 patients had to be kept in bed because of injuries other than the fractures. Many patients were discharged on crutches on the third day after the operation. When the operation took place on the day following admission and the patient was discharged on the third day after the operation, four days of hospitalization had been required, in contradistinction to the months spent in bed by patients given older methods of treatment. No patient whose condition was not complicated by other injuries had a total stay of more than seventeen days in the hospital.

On their discharge from the hospital on crutches, patients are instructed to return to the outpatient department at monthly intervals, unless discomfort or other symptoms bring them in sooner. At these periods check-up roentgenograms are taken to show the progress of the fracture.

In this series of cases the oldest patient was 66 years old and the youngest 7 years old. The fractures in patients from 20 to 40 years old

showed no more rapidity in healing than did those in patients past 40. The fractures in children from 7 to 16 years old, of which there were 3, required approximately one-half the healing time which was required by those in patients more than 20 years old. The average length of time elapsing after the original injury before roentgen evidence of healing was first seen was eighty-one days. The average postoperative period before evidences of healing first appeared through the cast was seventy-

Fig. 4.—This patient was up, on crutches, on the fourth day after the operation, five days after the original injury.

two days. It is to be noted that roentgen evidence of callus is obscured by plaster of paris. The pins were removed when roentgen evidence became positive for maintenance of the position of the bones without the pins. An average healing period of one hundred and twelve days after the operation was found to be satisfactory in this series. A short plaster of paris leg dressing extending from just below the knee to the toes, including a walking iron, was applied at the end of this period.

In the series there were 20 cases of sufficient duration to demonstrate functional union clinically and roentgenologically. The average length of time from the time of injury to the time of functional union was one hundred and fifty days, or one hundred and forty-three days

Fig. 5.—Tibiofibular fracture. Note that the pins have been removed and that union is satisfactory one hundred and sixteen days after the original injury. This fracture was kept in a plaster of paris dressing for an additional two months.

after insertion of the pins (fig. 5). However, the patient was kept in a cast and walking iron for an additional two months to insure firm union. The patients were free from all methods of immobilization at the end of an average period of seven months. Compound fractures

Fig. 6.—Final result, seven months after the original injury.

Fig. 7.—Leg after six or seven months' treatment for fracture of both bones. Note the pin scars above and below.

required on an average thirty-six days more to arrive at functional union. In only 1 case did any infection occur about the site of a pin. This was controlled by removal of the pin. There were no sequelae. In the 23 cases in which we have been able to follow the course through at least the first four months after the occurrence of the accident, there has been no instance of nonunion and but 1 instance of delayed union. The fracture in this single case was badly comminuted and compounded and was also infected originally. At present satisfactory union is slowly taking place. Satisfactory alinement and apposition were gained in each case. In no case was there slipping or angulation of the fragments following approximation with pin traction and immobilization by the original cast.

CONCLUSIONS

1. Fractures of the leg involving the tibia and the fibula together, not involving a joint, are best treated by the double steel pin traction in plaster of paris.

2. The compounding of a fracture is not an indication for a substitute method or for postponement of the use of this method, although a longer time is necessary for healing.

3. The period of hospitalization is minimal, and the patient is ambulatory early. The economic value of the treatment is obvious.

4. Satisfactory union may be expected within a period varying between seventy and ninety days.

5. The pins may be removed after about three and one-half or four months.

6. Functional union may be expected in five months, but we advise the continued use of the cast and the walking iron for an additional two months.

7. Nonunion has not occurred in this clinic with this type of management.

8. This method maintains anatomic approximation and insures a good functional result.

9. Satisfactory knee and ankle motion are noted after the removal of the cast.

10. The passive congestion associated with dependency of the leg and the irritation of the ends of the bones by the early use of graduated weight bearing are apparently definite stimuli to osteogenesis.

11. Patients are easily managed, happy and contented, and complain of very little pain or discomfort.

402 Genesee Bank Building.

SUPPURATIVE INTRATHORACIC THYROIDITIS

REPORT OF A CASE

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AND
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Although intrathoracic goiter is relatively frequent, its complication by suppuration is exceedingly rare. In a careful review of the literature, the only case found was one reported by Kalbfleisch [1] in 1920, in which the condition occurred in a young woman following a spontaneous abortion. Gas bacilli were present in the uterine secretions, although no local infection was observed. In the course of her illness, it was noted that a substernal goiter was present. In view of the septic course and local symptoms referable to the goiter, the diagnosis of either a complicating suppuration or a hemorrhage into the thyroid was considered. Operation was performed, and a suppurative intrathoracic goiter due to gas bacilli was found. Drainage was instituted, and recovery followed.

Acute thyroiditis has often been observed. Clute and Lahey [2] described two types: (1) simple thyroiditis, which is more common, usually follows infection of the upper respiratory tract and is generally resolved with conservative management; (2) suppurative thyroiditis, which is rarer and requires surgical intervention. Cochrane and Nowak [3] found acute thyroiditis in 0.5 per cent of all clinical cases of thyroid disease coming under their observation. The infection was usually metastatic, and the common organisms found were the staphylococcus, the streptococcus, the pneumococcus, the typhoid bacillus, the paratyphoid bacillus and the colon bacillus. In 1,985 patients with goiter treated surgically at the Cook County Hospital since 1929, there were only 4 with thyroiditis, an incidence of 0.2 per cent.

Statistics concerning the incidence of substernal or intrathoracic goiter vary considerably in the different clinics, probably because of

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From the Department of Pathology, Dr. R. H. Jaffé, Director, and the Medical Service of Dr. L. C. Gatewood, Cook County Hospital.

1. Kalbfleisch, K. W.: Metastatic Gas Bacillus Infection in a Retro-Sternal Cystic Goiter, Beitr. z. klin. Chir. **118**:342-362, 1920.

2. Clute, H. M., and Lahey, F. H.: Thyroiditis, Ann. Surg. **95**:493-498, 1932.

3. Cochrane, R. C., and Nowak, S. G.: Acute Thyroiditis, New England J. Med. **210**:935-942, 1934.

different modes of classification and terminology. Judd [4] found a substernal projection of the lobes of the thyroid in 50 per cent of patients presenting themselves for an operation on that gland, while the intrathoracic type was noted in less than 5 per cent. Goiter chiefly intrathoracic in location, without any cervical enlargement, was found in less than 1 per cent of the cases. Lahey [5] found no case of intrathoracic goiter in 4,363 cases of exophthalmic goiter or primary hyperthyroidism, but of 5,131 adenomatous goiters, 21 per cent extended into the superior mediastinum and the thorax. Of these goiters, 68 per cent reached nearly to the aortic arch, while 32 per cent extended to or below the arch. Sharer [6] found substernal goiter in 6 per cent of thyroidectomies and cited Higgins as stating that 10 per cent of goiters observed at operation were substernal and from 1 to 2 per cent intrathoracic. In 9,888 consecutive autopsies at the Cook County Hospital since 1929 there were found 1,222 nodular goiters. In this series only 3 true intrathoracic goiters were encountered, an incidence of 0.24 per cent.

Hemorrhage and cystic degeneration or other regressive changes are relatively common in substernal goiter, but suppuration is a rare complication. We are therefore reporting the following case of suppurative intrathoracic thyroiditis, the second case to be described in the literature.

REPORT OF CASE

History.—M. J., a Negress aged 27, entered the Cook County Hospital on Dec. 24, 1936. She was in such poor condition that a thorough history could not be obtained. However, it was learned that she had been generally well until eight days previously, when she noticed a pain in the right side of her chest, which continued, causing her to be short of breath. Cough was present since the onset, with expectoration, which at times was blood streaked. Chills were thought to have been present.

Physical examination revealed an obese Negress appearing acutely ill. The rectal temperature was 105 F., the pulse rate 154 and the respiratory rate 48. The essential findings were limited to the chest. There were dulness, bronchophony and tubular breathing in the lower lobe of the right lung and a friction rub over the right side of the chest. The heart showed no enlargement, and a systolic murmur was present over the apex. The abdomen was distended, and the liver was slightly enlarged and was tender. The diagnosis was lobar pneumonia of the lower lobe of the right lung.

The patient's condition improved gradually, the temperature falling by lysis. However, moderate lethargy remained, and in her fourth week in the hospital the oral temperature ranged between 99 and 101 F., the pulse rate between 100 and 120 and the respiratory rate between 20 and 30. The pulmonary symptoms were

4. Judd, E. S.: Intrathoracic Goiter, Internat. Clin. **1**:149, 1920.

5. Lahey, F. H., and Swinton, N. W.: Intrathoracic Goiter, Surg., Gynec. & Obst. **59**:627-637, 1934.

6. Sharer, R. F.: Substernal Thyroid, Am. J. Surg. **32**:56-62, 1936.

practically cleared up, and the patient had no complaints. A roentgenogram (fig. 1) showed a small area of infiltration in the lower third of the right lung, a widening of the superior mediastinal shadow and an elevation of the dome of the right side of the diaphragm (Dr. J. P. Bennett). Fluoroscopic examination and further studies were advised, but the patient's condition was too much weakened for these and she died suddenly on Jan. 24, 1937, five weeks after the onset of her illness. Slight icterus was noted.

Laboratory Findings.—The white blood count ranged between 22,000 and 24,800. The urine on several examinations was normal. Blood tests and Kahn tests of the spinal fluid gave negative results. The sputum showed no tubercle bacilli.

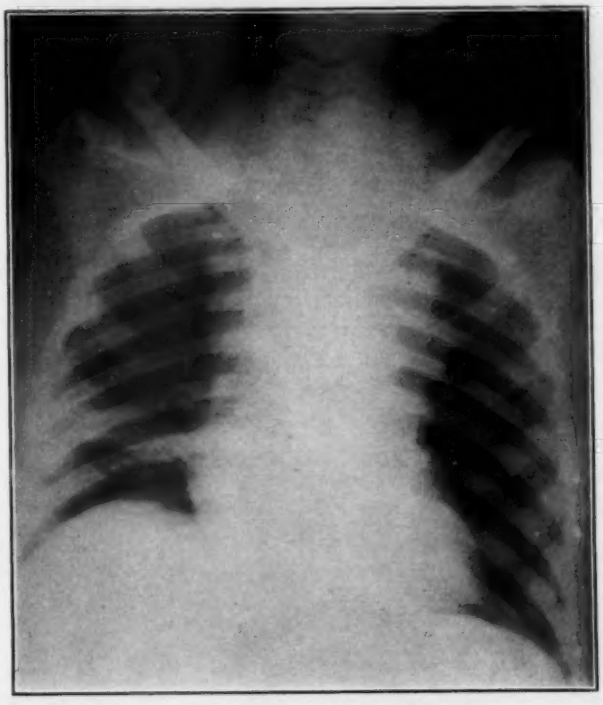

Fig. 1.—Anterior view showing marked widening of the mediastinum by the substernal nodose goiter.

The final clinical impression was that of lobar pneumonia which had been resolved except for a deep-seated abscess in the lower lobe of the right lung.

Abstract of Autopsy Report.—The thyroid gland was enlarged, and from the lower pole of its right lobe there extended a lobular mass through the superior thoracic aperture into the superior and anterior mediastinum, slightly compressing the trachea and the innominate veins (fig. 2). On the right side of the trachea the mass, together with the thyroid, measured 17 by 6 by 7 cm., while on the left side the mass measured 16.5 by 7 by 6 cm. The left lobe of the thyroid could be separated from the mass. It measured 8 by 5 by 5.5 cm. in diameter and contained an oval, pale yellow-gray node 5 by 4 cm. in diameter. The right lobe of the thyroid measured 8 by 3 by 2 cm. When sectioned it appeared light gray-

Fig. 2.—Anterior view showing nodular masses of the right lobe of the thyroid extending into the superior and anterior mediastinum and compressing the trachea. *L*, indicates lungs; *H*, heart.

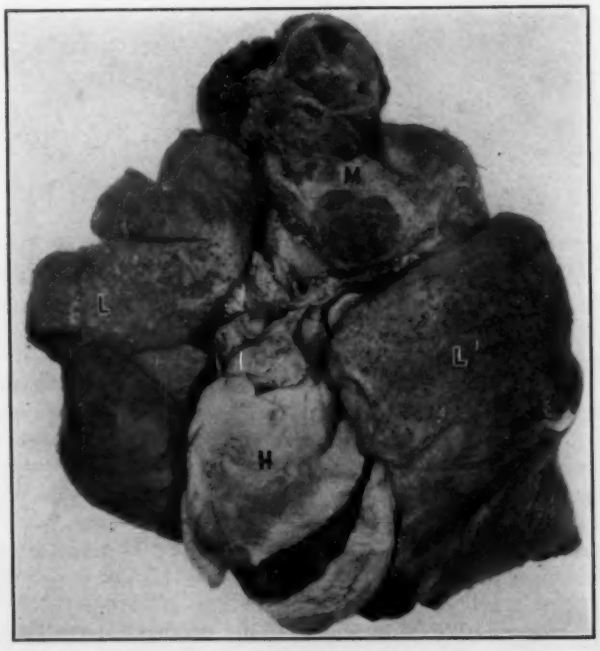

Fig. 3.—Posterior view showing the nodose goiter in the posterior mediastinum (*M*) and its relation to the lungs (*L*) and the heart (*H*).

brown, was coarsely granular and was rich in colloid material. The mediastinal mass fused with the isthmic portion of the thyroid and was composed of multiple moderately firm spherical and ovoid nodes which varied from 10 to 35 mm. in diameter (fig. 3). The nodes were pale gray-tan and mottled with dark purple-red patches of diameters up to 4 mm. Between the nodes, especially on the right side, there were several cavities from 6 to 11 mm. in diameter filled with a creamy light yellow-gray pus. These cavities were surrounded by firm gray-white tissue.

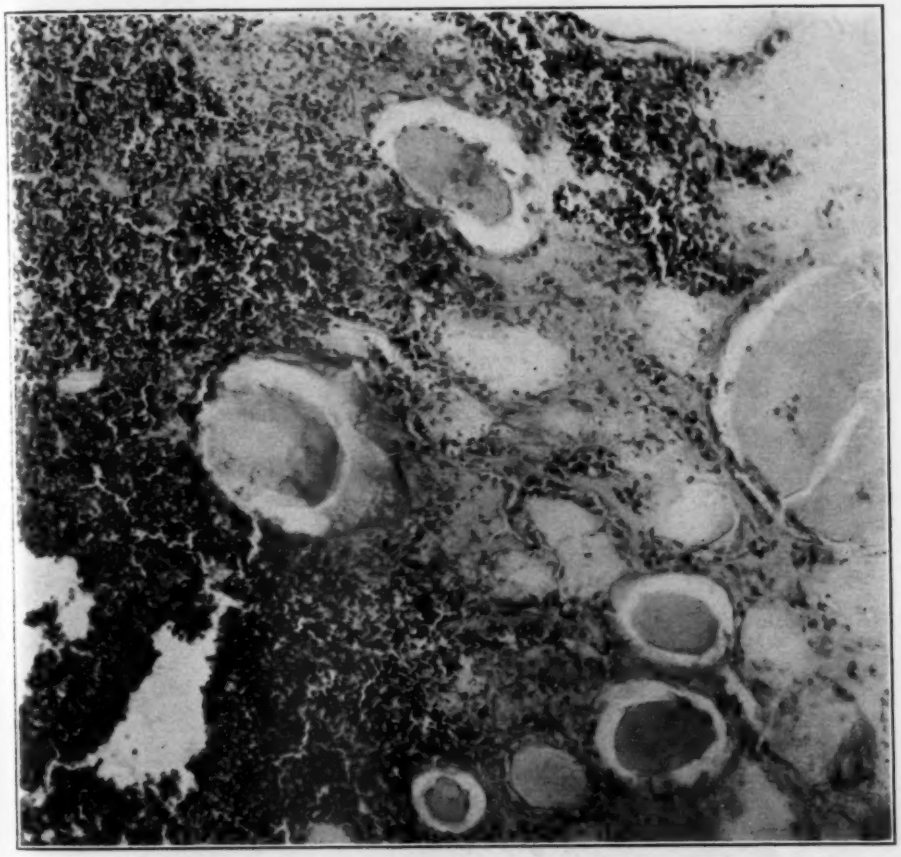

Fig. 4.—Photomicrograph of the wall of the abscess associated with the retrosternal goiter, showing degenerated follicles and the stroma densely infiltrated by polymorphonuclear leukocytes. Magnification, × 150.

In the left pleural cavity there were focal fibrous adhesions about the lateral aspect of the upper lobe of the lung, while the right side was partially obliterated by loose fibrous adhesions. Both lungs were moderately distended and were crepitant. The surfaces revealed by section were purple-gray and moist. The pneumonic process had been completely resolved.

There was a slight hypertrophy of the heart, which weighed 360 Gm.

The spleen was enlarged and soft and weighed 190 Gm.

Microscopic Findings.—The mediastinal mass consisted of numerous nodes separated by strands of dense connective tissue. The nodes varied in size; the

smaller ones were composed of small and medium-sized follicles filled with a pale-stained, often vacuolated colloid material and lined by a cuboid or low cylindric epithelium. In the larger nodes there were large follicles lined for the most part by a low cuboid epithelium. In places the structure was interrupted by dense accumulations of plasma cells and round cells which infiltrated the septums and extended also into the nodes. Mixed with the plasma cells were a moderate

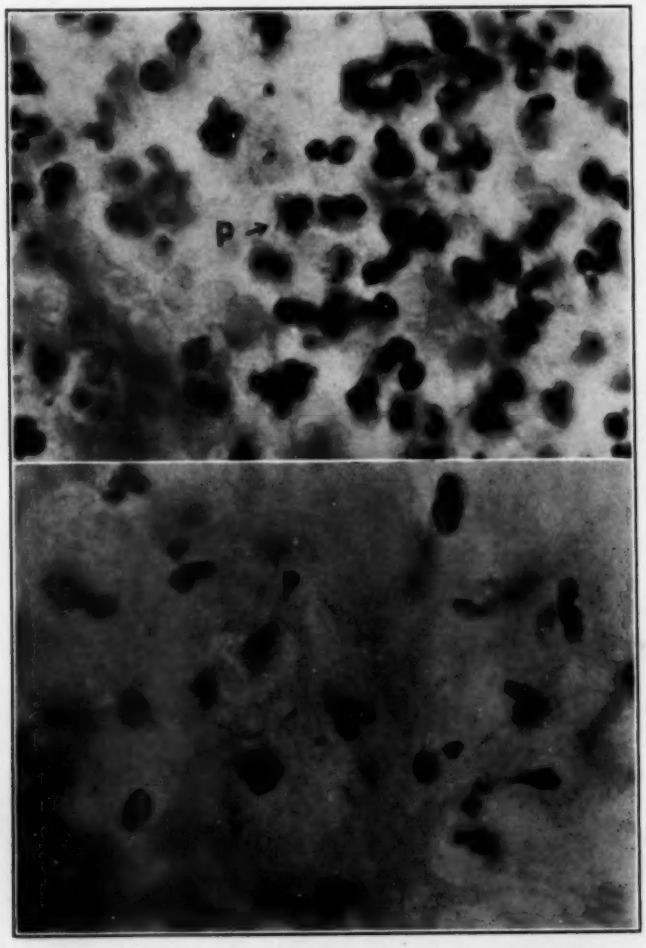

Fig. 5.—Photomicrograph showing pneumococci in the wall of the abscess cavity. Gram-Weigert stain; magnification, × 1,000.

number of iron-filled histiocytes. There were focal deposits of lime salts. In places, recent extravasations of blood were seen. In the periphery of the small nodes, accumulations of pus cells were present, but the most marked suppurative changes were encountered in the large nodes and seemed to select particularly those areas which were the sites of preceding regressive processes (fig. 4). In these areas the follicles were necrotic, the stroma was hyalinized and there were coarse calcium deposits as well as deposits from old and recent hemorrhages.

In the midst of the necrotic tissue were dense accumulations of pus cells with liquefaction of the follicles that were involved.

The left lobe of the thyroid gland was chiefly occupied by nodes composed of large colloid-filled follicles with focal areas of epithelial hyperplasia and proliferation. The stroma contained nets of iron-filled histiocytes. In the right lobe of the thyroid there were nodules made up of small and medium-sized follicles lined by cuboid or low cylindric epithelium.

Gram-Weigert staining of the sections from the substernal mass revealed numerous free lancet-shaped gram-positive diplococci (fig. 5).

Bacterioscopic Examination.—In smears taken from the cavities of the substernal portion of the thyroid, numerous lancet-shaped, gram-positive diplococci and clumps of pus cells were found.

Anatomic Diagnosis.—The diagnosis was multiple abscesses in a nodose, retrosternal goiter; bilateral nodose goiter; compression of the trachea and the innominate veins by the retrosternal mass; slight hypertrophy of the heart; passive congestion of the lungs; infectious hyperplasia of the spleen, and acute glomerulitis.

COMMENT

From the history, autopsic observations and bacteriologic examination of the pus, it may be assumed that the retrosternal thyroiditis succeeded lobar pneumonia which at the time of the autopsy had been completely resolved. The lack of elasticity and the friability of the lower lobe of the right lung were characteristic of the condition of the lung that followed the resorption of a pneumonic exudate. Since the retrosternal goiter was adherent to the mediastinal pleura, one may assume that the infection spread to the thyroid by continuity.

In cases of suppurative thyroiditis the route of infection is usually hematogenous. Such a hematogenous abscess of the thyroid has been observed by us in a case of endocarditis caused by Streptococcus viridans.

Clinically, the main symptoms of substernal goiter are produced by the compression of the mediastinal structures, that is, dyspnea, cyanosis of the face and neck, choking spells and dysphagia. When in addition septic manifestations are present, one may suspect a complicating infection of the goiter. In the absence of evidence of compression, roentgenologic examination may furnish the clue as to the focus of infection, namely, the marked widening of the mediastinal shadow.

SUMMARY

A case of suppuration in an intrathoracic nodose goiter is presented. The suppuration was apparently secondary to lobar pneumonia which at the time of the patient's death had been completely resolved. The mode of infection of the retrosternal goiter was probably by continuity, since the goiter was adherent to the mediastinal pleura, which in turn was adherent to the lung. Among 1,222 nodose goiters observed at autopsy, 3 intrathoracic goiters were encountered.

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POLYOSTOTIC FIBROUS DYSPLASIA

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My attention was recently drawn by Dr. Henry L. Jaffe, director of the laboratories at the Hospital for Joint Diseases, to a number of cases (4 from this and 4 more from other hospitals from which pathologic material was studied) presenting multiple osseous lesions and having sufficient features in common to differentiate them as a distinct clinical entity. The condition is designated as polyostotic fibrous dysplasia, for reasons to be indicated presently.

DEFINITION AND NOMENCLATURE

The name "polyostotic fibrous dysplasia" is being used in the Hospital for Joint Diseases to designate a skeletal developmental anomaly affecting several or many bones with predominantly unilateral involvement. The involved bones show filling of their medullary cavities by gritty, grayish white fibrous tissue containing trabeculae of newly formed primitive bone. The condition apparently results from perverted activity of the specific bone-forming mesenchyme. It manifests itself in childhood or early adult life and evolves slowly, pursuing a protracted clinical course characterized by pain, deformity and a tendency to pathologic fracture of the affected bones.

A number of other cases which I regard as instances of the disease have been described in the literature under a bewildering variety of titles, such as, for instance, osteodystrophia fibrosa unilateralis, unilateral polyostotic osteitis fibrosa, unilateral Recklinghausen's disease, osteodystrophia fibrosa cystica generalisata limited to one side of the body, focal osteitis fibrosa, osteitis fibrosa in multiple foci, osteitis fibrosa with formation of hyaline cartilage and osteitis fibrosa disseminata.[1]

From the Laboratory Division, Hospital for Joint Diseases.

1. A great deal of confusion exists with regard to the nomenclature of skeletal diseases, inherited largely from early investigators. The term "osteitis fibrosa" was employed in the German literature until comparatively recently to include the osseous lesions of hyperparathyroidism, Paget's disease, giant cell tumors, cysts of the bone and localized osteitis fibrosa. The term "fibrous osteodystrophy" has been applied to the same miscellaneous, pathologically unrelated group of osseous diseases, and in fact is still used in the *Index Medicus* synonymously with osteitis fibrosa. The terms nongeneralized fibrous osteodystrophy and unilateral fibrous osteodystrophy, introduced by some authors to obviate the confusion of the

(Footnote continued on next page)

The titles purporting to show the relation of the condition to Recklinghausen's disease of the bone are obviously erroneous, while those emphasizing the unilateral distribution of the affected bones are misleading. The disorder, it is true, shows some tendency to unilaterality. However, in severe forms there is often involvement of the bones on the opposite side and also of the skull and of the vertebral column. Osteitis fibrosa in multiple foci and other similar names qualifying osteitis fibrosa in one way or another are accurate as far as they go but fail to convey an adequate impression of the pathogenesis of the disease. It may be seen that no one of the titles hitherto suggested is altogether satisfactory. I have therefore felt obliged, despite an overburdened nomenclature, to propose the name polyostotic fibrous dysplasia to designate the disease in question.

CLINICAL FEATURES

Incidence According to Age and Sex.—The youngest patient in my own group was 19 years of age; the oldest, 47. The rest of the patients were women in their early twenties. However, in regard to incidence by age, if one considers cases already reported in the literature, the impression is gained that severe involvement may become clinically manifest in adolescence or even as early as childhood. By the time patients seek treatment, the disease as a rule has already existed for some years, as evidenced by clinical symptoms dating back to the first and second decades of life. In reviewing the histories of my own cases I note that the average age at the onset of symptoms was approximately 10 years. The disease apparently has a distinct tendency to occur in women. The ratio of women to men among the patients in my material and in that of other observers is at least 3 to 1.

Clinical Manifestations.—The common clinical signs and symptoms of polyostotic fibrous dysplasia are pathologic fracture, limp, pain and deformity of the affected limb. Pathologic fracture, especially of the femur and occasionally of the humerus, radius or wrist, is perhaps the most frequent symptom. Pain in the hip, also referred to as soreness or stiffness, is another common complaint among those suffering from this disease. Patients may seek treatment also because of shortening of a lower extremity, coxa vara and bowing of the femur, distressing pain in the tibia and occasionally deformity of an upper extremity.

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condition under discussion with generalized osteitis fibrosa cystica, are open to the same objection. It seems to me that the situation would be much clarified if giant cell tumors, simple cysts of the bone of undetermined origin and localized osteitis fibrosa (i. e., fibrosis of the marrow) due to whatever cause were regarded as different entities until more is known of their pathogenesis and the name polyostotic fibrous dysplasia introduced for the clinical entity discussed in this paper.

The long duration of symptoms is an indication of the chronicity of the disease and the slow progression of its osseous lesions. The known duration of symptoms in my series of patients was more than twenty years on the average and in 1 instance as much as thirty-six years.

Precocious menstruation in children suffering from severe forms of the disease has been described by Goldhamer,[2] Borak and Doll [3] and recently by Albright and associates.[4] Goldhamer felt that this manifestation of pubertas praecox might in some way be attributed to involvement of the base of the skull. Its significance, however, remains a matter of conjecture.

The presence of hyperpigmentation of certain areas of the skin, apparently due to excessive melanin content, has also been mentioned by Goldhamer and particularly stressed by Albright and his associates as another feature occasionally found in association with the osseous lesions. The precise significance of hyperpigmentation in relation to the osseous lesions is impossible to decide in the present state of medical knowledge. In none of the cases which I have described was excessive pigmentation of the skin noted.

DISTRIBUTION OF OSSEOUS LESIONS

The osseous lesions tend to be exclusively or predominantly unilateral, apparently affecting either side without preference. However, bilateral lesions also occur, albeit in a minority of cases. In my material the lesions demonstrable on roentgen examination of the skeleton were entirely unilateral in 3 instances, predominantly unilateral in 2 and bilateral in 1. This is essentially in accord with the composite experience of other observers. The bones of the lower extremity, i. e., the femur and the tibia, according to my experience, are affected much more often than are the humerus and the radius. Combined involvement of lower and upper extremities is not infrequent.

The long bones of the extremities are most often affected and in the following order of frequency: femur, tibia, humerus and radius. Next in order of frequency are the bones of the skull (cranial vault

2. Goldhamer, K.: Osteodystrophia fibrosa unilateralis (kombiniert mit Pubertas praecox und mit gleichseitigen osteosklerotischen Veränderungen der Schädelbasis), Wien. klin. Wchnschr. **47**:218, 1934; Fortschr. a. d. Geb. d. Röntgenstrahlen **49**:456, 1934.

3. Borak, J., and Doll, B.: Halbseitige Recklinghausensche Knochenkrankheit mit Pubertas praecox, Wien. klin. Wchnschr. **47**:540, 1934.

4. Albright, F.; Butler, A. M.; Hampton, A. O., and Smith, P.: Syndrome Characterized by Osteitis Fibrosa Disseminata, Areas of Pigmentation and Endocrine Dysfunction, with Precocious Puberty in Females, New England J. Med. **216**:727, 1937.

and jaw bones), pelvic bones, ribs and phalanges. Lesions have been noted roentgenographically also in the vertebrae, scapula, clavicle, metacarpals, fibula and other bones. My experience of necropsies on subjects who had had Paget's disease, multiple myeloma, Gaucher's disease and other generalized osseous diseases has indicated that roentgen examination does not begin to reveal the actual extent of involvement of the skeleton. That would probably be just as true for fibrous dysplasia. I realize full well, therefore, that my impressions as to the frequency and extent of the lesions in polyostotic fibrous dysplasia are only as accurate as the roentgen examination of the skeleton is reliable and thorough. In a general way, however, they indicate the relative frequency of at least the conspicuous or well developed osseous lesions.

DIAGNOSIS

Interpretation of Roentgenographic Observations.—The objective features seen in roentgenograms of bones affected with polyostotic fibrous dysplasia may be summarized as follows: (1) broadening or expansion of the bone; (2) thinning of the cortex; (3) characteristic rarefied and apparently trabeculated appearance, usually erroneously interpreted as indicating "cystic disease," and (4) secondary deformities of affected bones. In the lower extremity, these are principally coxa vara and bowing of the femur or of the tibia. When involvement is severe, the upper end of the femur may be deformed and the neck shortened. Pathologic fracture of the neck of the femur is a frequent development. With severe involvement of the upper extremity, there may be bowing and even spontaneous fracture of the humerus. When the process is well developed, the roentgenogram is usually characteristic, especially if the skeleton as a whole is examined (or at least both femurs, both tibias, the pelvis, the ribs and the skull).

The commonest error in interpreting roentgenograms of the lesions of polyostotic fibrous dysplasia is to construe them as indicating "cystic disease." This explains why in many cases the condition is clinically misdiagnosed as osteitis fibrosa cystica or Recklinghausen's disease of the bone. When the involvement is predominantly unilateral, as it frequently is, the diagnosis of unilateral Recklinghausen's disease has occasionally been made. If attention is focused on a single bone, without roentgen examination of the remainder of the skeleton, an erroneous diagnosis of giant cell tumor, localized cyst of the bone or even enchondroma may be entertained. In point of fact, the involved bones contain no cysts, and biopsy demonstrates beyond a shadow of a doubt that the peculiar appearance of the lesions is due to replacement of the spongy bone and filling of the medullary cavity by fibrous tissue containing spicules of poorly calcified primitive bone. The trabeculated

appearance probably reflects irregularities in the extent of erosion of the inner surface of the cortical bone (fig. 1).

Chemical Findings in the Blood.—Estimations of serum calcium were done in the laboratory in 5 cases in which the diagnosis of polyostotic fibrous dysplasia was confirmed by biopsy. The values ranged between 9.8 and 11.0 mg. per hundred cubic centimeters. Values for serum calcium ranging between 11.0 and 11.6 mg. have been noted by other observers also (Borak and Doll,[3] Albright and associates [4] and Freund and Meffert [5]). Apparently, then, the concentration of calcium in the serum may approach the upper limit of the normal range or even be slightly elevated.

Estimations of serum phosphorus show no significant deviation. Nor do any of the other chemical constituents of the blood, with the exception of phosphatase, show any appreciable alteration in their relative concentration.

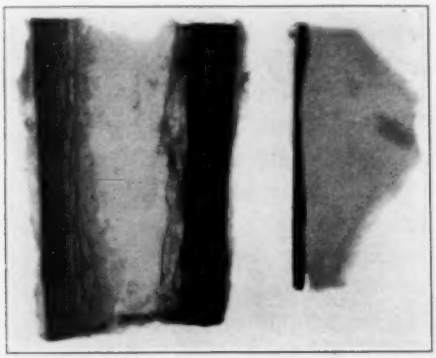

Fig. 1.—Roentgenogram of a portion of a diseased tibia (specimen taken for biopsy in case 2), illustrating the marked thinning of the cortex, the absence of deposition of periosteal bone, the endosteal erosion of the cortex and the filling of the medullary cavity by a homogeneous fibrous tissue containing tiny spicules of bone. Compare with the normal tibia from a woman, on the left.

The phosphatase content, however, was notably increased in 3 of my 4 cases, being 17, 18 and 22 units, respectively, when determined by Bodansky's method (as compared with the normal upper limit for adults of 4 units). Increased serum phosphatase has been noted by other observers also (Albright and associates [4] and Freund and Meffert [5]). The increased values for phosphatase in fibrous dysplasia afford additional support for the hypothesis of Bodansky and Jaffe [6] that the

5. Freund, E., and Meffert, C. B.: On the Different Forms of Non-Generalized Fibrous Osteodystrophy, Surg., Gynec. & Obst. **62**:541, 1936.

6. Bodansky, A., and Jaffe, H. L.: Phosphatase Studies: III. Serum Phosphatase in Diseases of the Bone; Interpretation and Significance, Arch. Int. Med. **54**:88 (July) 1934.

activity of the enzyme phosphatase is proportional to the stimulus to the formation of new bone. In this connection, it is interesting to note that in the case in my series in which the value for phosphatase was the lowest (4.1 units) biopsy revealed the least tendency to or capacity for reparative formation of new bone within the fibrous tissue of the lesion, in contradistinction to 2 other cases, in which there was a considerably greater osteogenic tendency, associated with values for phosphatase of 22 and 17 units (Bodansky), respectively. In evaluating the chemical observations of the blood, then, one should remember that the calcium content may be normal or slightly increased and that the proportion of phosphatase is frequently elevated.

REPORT OF CASES

In order that the clinicians encountering patients with polyostotic fibrous dysplasia—and the condition is by no means infrequent—may be enabled more readily to recognize it as a clinical entity, the following representative cases with illustrative photomicrographs and roentgenograms are reported.

CASE 1.—The patient was a married woman, 19 years old, who was admitted to the hospital to the service of Dr. Eising on Feb. 24, 1926, complaining of pain in the right hip and a limp. She dated her illness back to an alleged fall at the age of 8, for which she was treated for several months at another hospital. Whether the patient sustained a fracture at that time was not stated. Physical examination revealed limitation of abduction and internal rotation and 1 inch (2.5 cm.) of shortening of the right lower extremity. The clinical laboratory findings were of no particular significance. The tentative clinical diagnosis on admission was traumatic coxa vara and "cystic condition" of the right femur.

Roentgenograms of the right femur disclosed broadening and rarefaction of the upper two thirds. The cortex of the bone was thin but not perforated. There was a focal area of thickening of the cortical bone in the upper third of the femur, which suggested the site of an old healed fracture. The osseous structure had a hazy, ground glass, trabeculated appearance. The upper third of the femur was markedly deformed; the neck was shortened and the position was that of a coxa vara. The lower end of the femur also showed a number of focal rarefied areas. Roentgenograms of the ribs showed expansion of several, notably the ninth and the eleventh, on both sides. Roentgen examination of the bones of the upper extremity, the left femur, the vertebral column and the skull showed no demonstrable osseous defects. Roentgen examination of the femur and tibia nine years later (fig. 2) showed that the osseous lesions had persisted and, if anything, had progressed. There were distinct rarefied and apparently trabeculated lesions not only in the femur but in the upper end of the tibia.

Biopsy of a portion of the ninth right rib was performed for diagnosis. The cortex was extremely thinned, and the medullary cavity was filled with a firm grayish white fibrous tissue. Microscopic examination showed small trabeculae of primitive, poorly calcified new bone irregularly dispersed within the fibrous tissue (fig. 3). Study of the sections in conjunction with the history and the roentgenographic observations indicated that this was a case of polyostotic fibrous dysplasia. The

Fig. 2.—*A*, roentgenogram of the right femur in case 1. *B*, roentgenogram of the lower third of the right femur and the upper end of the tibia in case 1.

Fig. 3.—Biopsy specimen of a portion of the ninth rib in case 1, cross section, showing the absence of periosteal deposition of new bone, the thinning of the cortex with erosion of the endosteal surface and the medullary cavity filled solidly with fibrous tissue containing small spicules of calcified immature bone (× 15).

surgical wound healed uneventfully, and the patient was treated with ultraviolet rays. For the next several years, however, she complained of persistent soreness in the right thigh, and in 1930 she was operated on at another hospital, where an autogenous tibial bone graft was placed in the right femur (fig. 2 *A*). Subsequently she was obliged to wear a brace. The follow-up record from the same institution indicates that in 1934 the patient received a series of six roentgen exposures to the region of the parathyroid glands and that in 1936 bilateral exploration of these glands was performed but no enlarged ones were observed.

Case 2.—The patient, a woman 22 years old, was admitted to the hospital, to the service of Dr. Herman Frauenthal, on July 17, 1935, because of a pathologic fracture of the neck of the right femur. The patient also presented a limp, which she said was first noted at the age of 5. No record of familial incidence was elicited. The past history was irrelevant except for a "traumatic fracture" of the right ankle three years earlier. Physical examination revealed evidences of

Fig. 4.—Roentgenogram of the pelvis and upper portions of the femurs in case 2, showing the involvement of the right femur and ilium, and of the pubis and ischium on both sides.

pathologic fracture of the neck of the femur with upward displacement of the shaft of about ¾ inch (2 cm.). Relative thinning of the right lower extremity was also noted. Chemical findings in the blood were as follows: calcium 10.0 mg., phosphorus 2.0 mg. and phosphatase 22.6 units (Bodansky). Subsequent estimations gave values of 9.8 mg., 2.2 mg. and 18.1 units, respectively. The other data revealed in the laboratory were of no particular significance. The tentative clinical diagnosis on admission was "cystic disease" of the bones. The possibility of Hand-Schüller-Christian disease was considered by some because of the involvement of the skull (fig. 7) and possible slight exophthalmos.

Roentgen examination (figs. 4, 5, 6, 7 and 8) disclosed osseous lesions in the right femur and tibia, in the pubis and ischium on both sides and also in the skull. There was a pathologic fracture through the neck of the right femur with upward displacement of the shaft and the production of a coxa vara. The entire right

Fig. 5.—Roentgenogram of the lower third of the femurs and upper ends of tibias in case 2. Note the shortening of the affected extremity.

Fig. 6.—Roentgenogram of the tibias in case 2.

Fig. 7.—Roentgenogram of the skull in case 2. The arrows indicate the osseous defects.

Fig. 8.—Roentgenogram of the vertebral column and ribs in case 2, showing lesions in the seventh and eighth ribs.

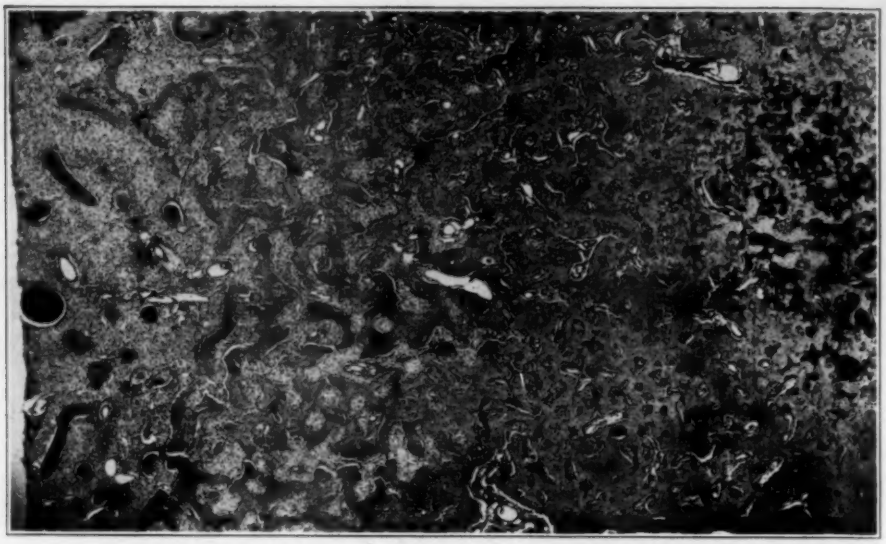

Fig. 9.—Biopsy specimen of the right tibia in case 2. Note the advanced lesion showing considerable deposition of fiber bone in the fibrous matrix and irregular focal calcification (× 15).

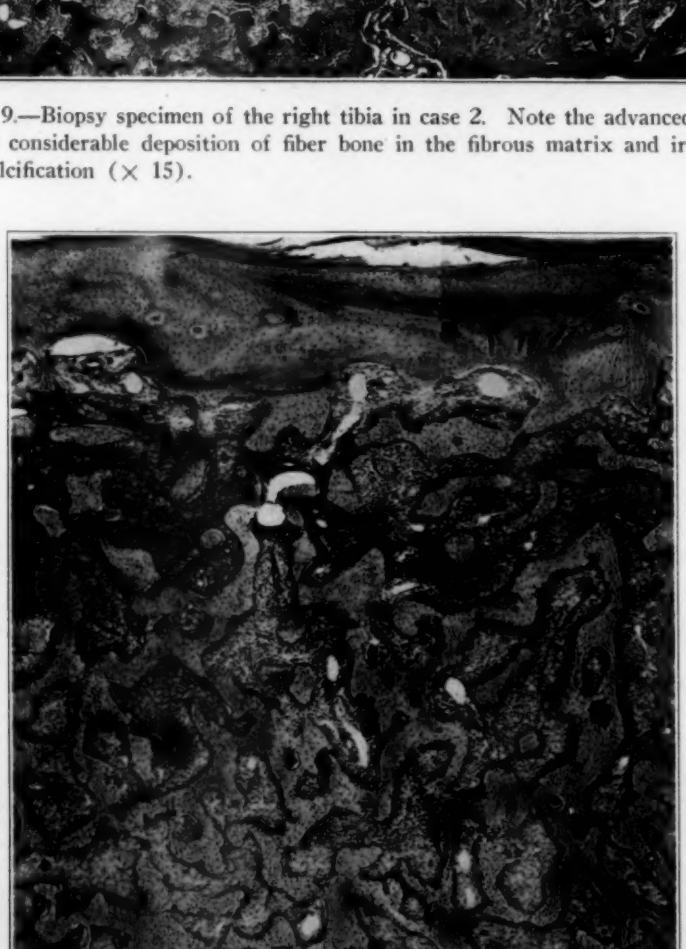

Fig. 10.—Biopsy specimen of the right tibia in case 2, cross section, showing absence of periosteal reaction, the thinning of the cortex with endosteal resorption and the replacement of the substantia spongiosa and filling of the medullary cavity by fibrous tissue containing irregular trabeculae of fibrous bone (× 25).

femur and tibia showed pronounced broadening and rarefaction. The lower end of the femur in particular presented a clublike expansion. The cortex was thinned and slightly irregular. The osseous structure had a mottled, porotic appearance. The contiguous parts of the right ilium, ischium and pubis, as well as the ischium and pubis on the opposite side, were similarly involved. Lesions were noted also in the seventh and eighth ribs and in the eighth dorsal vertebra. A roentgenogram of the skull disclosed two large, ovoid, rarefied osseous defects in the upper parietal and occipital regions. There was also a large blister-like lesion in the occipital bone, which had expanded the outer table of the skull (fig. 8).

Biopsy of the right tibia was performed for diagnosis. The cortex was found to be reduced to a thickness of approximately 1 mm. and the medullar cavity filled with a peculiar type of solid, whitish spongy tissue. Microscopic examination (figs. 9 and 10) showed fibrous tissue in which large trabeculae of immature, poorly calcified new bone had been deposited by osseous metaplasia. The histologic sections, in conjunction with the clinical history and the roentgenographic observations, indicate that this is a case of polyostotic fibrous dysplasia in an active stage. Healing of the wound inflicted in removal of tissue for biopsy was slow but uneventful, and the patient was discharged about two weeks later, walking on crutches. She left New York subsequently, but a personal communication two years later was to the effect that healing of the bone was delayed for more than six months and that pain in the leg had persisted. A local surgeon had suggested exploration of the neck for parathyroid tumor (a common error!) but the patient was advised not to accept this counsel.

CASE 3.—The patient, a woman 20 years of age, was admitted to the hospital on Nov. 3, 1936, to the service of Dr. Kleinberg, complaining of a limp, which had been manifest since she was 10 years old. The patient also complained of stiffness of the left hip and pain on exertion or exposure to cold. Physical examination disclosed a slight limp on the left side, marked posterior curvature of the vertebral column and mild anterior and marked outward bowing of the upper third of the left thigh. Complete restriction of abduction was noted and also marked limitation of inward rotation. There was tenderness in response to pressure over the lesser trochanter. Significant chemical findings in the blood were: calcium 10.4 mg., phosphorus 3.4 mg. and phosphatase 17.1 units (Bodansky). The other laboratory findings, including the value for serum proteins, were within the normal range of variation. The tentative clinical diagnosis on admission was "cystic disease" of the bones. Other possibilities suggested were Ollier's disease and giant cell tumor.

Roentgenograms (figs. 11 and 12) disclosed extensive involvement of the left ilium, femur and tibia. These bones had an expanded, porotic and trabeculated appearance. There was distinct cortical thinning without any perforation. The changes were most pronounced in the ilium and the upper third of the femur but were also evident in the lower third of the femur and in the tibia. Since a specimen of tissue taken for biopsy by means of a punch proved insufficient for diagnosis, it was decided to attempt collapse of the expanded upper end of the femur. The cortex was found to be much thinned out and the medullar cavity filled with a whitish, fibrous, gritty tissue of rubbery consistency. Sections (figs. 13 and 14) showed replacement of the spongy bone and filling of the medullar space by a fibrous tissue made up of spindle cells, in which scattered trabeculae of osteoid and atypically calcified fiber bone had been formed by osseous metaplasia. There was also a number of small focal islands of hyaline cartilage within the

Fig. 11.—Roentgenogram of the pelvis and the upper portions of the femurs in case 3.

Fig. 12.—*A*, roentgenogram of the lower end of the left femur in case 3. *B*, roentgenogram of the left tibia in case 3.

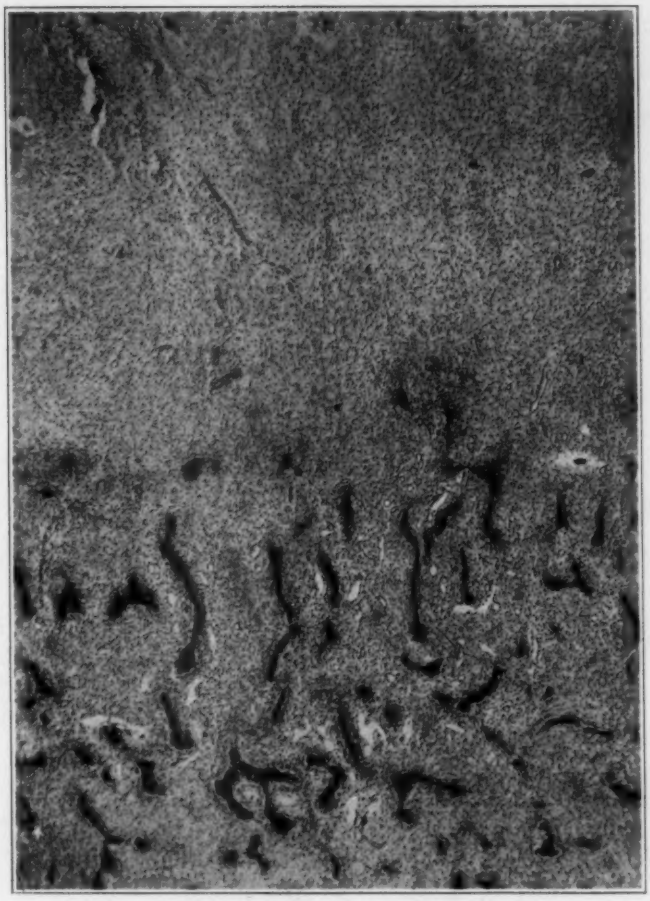

Fig. 13.—Fibrous lesion in case 3, showing an area of relatively sparse deposition of new bone (× 15).

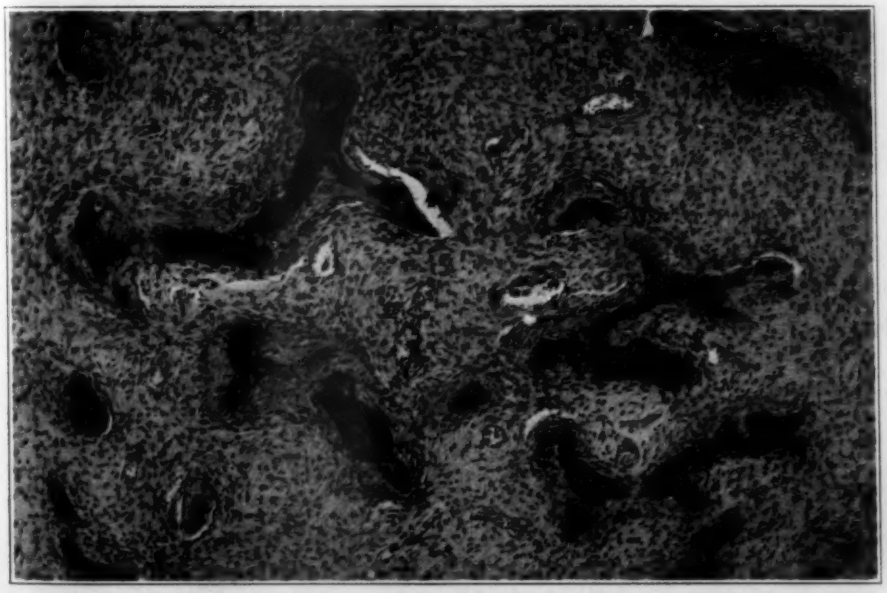

Fig. 14.—Higher magnification of the fibrous lesion seen in figure 13. Note the formation of trabeculae of fiber bone by osseous metaplasia, the irregular atypical calcification and the relatively small number of blood vessels (× 45).

fibrous tissue (fig. 15). Biopsy, in conjunction with the clinical and roentgenographic observations, indicated that this also was a case of polyostotic fibrous dysplasia.

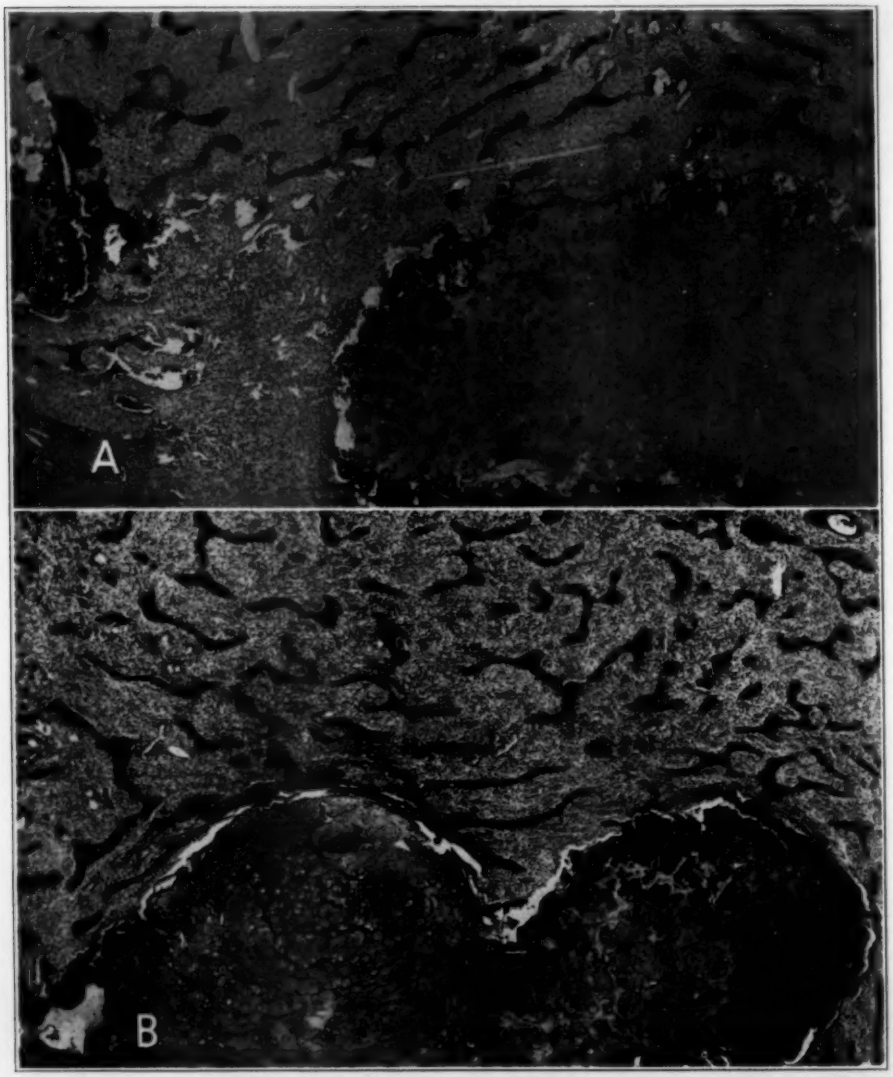

Fig. 15.—Fibrous lesion in the left femur in case 3. *A*, islands of hyaline cartilage undergoing calcification, showing a few giant cells around the capillary extravasation (× 15). *B*, focal area of hyaline cartilage calcified at its periphery (× 15).

Convalescence following the operative procedure was uneventful, and the patient was discharged after three weeks. Several months later she was readmitted because of persistent pain, and a second curettage of the femur was performed. Biopsy

confirmed the previous observations. Chemical analysis of the blood at the time of the second admission gave the following values: calcium 10.9 mg., phosphorus 3.9 mg. and phosphatase 12.4 units (Bodansky). Moderate secondary anemia, which required transfusion, developed after operation. This may be a partial explanation for the lowering of the phosphatase content of the serum. The patient was discharged after several weeks' convalescence.

CASE 4.—The patient, a man aged 47, was admitted to the hospital Oct. 9, 1933, to the service of Dr. Finkelstein, complaining of severe pain in the right tibia. He gave a history of deformity of the right leg dating back to childhood, because of which he was rejected for military service as a youth. He had been referred by the outpatient department for further study, with the suggestion that he might have Paget's disease. Physical examination disclosed marked anterior bowing of the tibia and atrophy of the muscles of the right lower extremity. It was incidentally observed that he presented tremors of the type seen in Parkinson's disease Chemical findings in the blood were: calcium 10.0 mg., phosphorus 2.7 mg., phosphatase 4.1 units (Bodansky). The other laboratory data were of no particular significance.

Roentgenograms (fig. 16) disclosed pronounced cortical thinning and expansion of the distal third of the shaft of the femur. The osseous structure had a mottled, porotic appearance, with focal areas of diminished density. The most extensive changes were observed in the tibia, which showed medullary expansion, irregular cortical thinning and multiple rarefied osseous defects within the shaft. There was also anterior bowing of the tibia. Roentgen examination of the vertebral column and skull showed no osseous lesions. Biopsy of the right tibia was performed for diagnosis. The cortex was thin and showed evidence of resorption from the medullary side. There was complete absence of periosteal deposition of bone. The medullar cavity was occupied by a gritty, spongy fibrous tissue. Sections (fig. 17) showed small trabeculae of atypically calcified primitive bone irregularly dispersed within the fibrous tissue. Biopsy in conjunction with the clinical and roentgenographic observations indicated that this was a clearcut case of polyostotic fibrous dysplasia. The operative wound healed uneventfully. The severe pain in the tibia was unrelieved, however. In May 1936 the patient was treated in another hospital for erysipelas. Roentgen examination of his skeleton confirmed the presence of the osseous lesions previously noted.

PATHOLOGIC PICTURE

There are few comprehensive and well illustrated pathologic descriptions in the literature dealing with the subject. As patients suffering from polyostotic fibrous dysplasia do not ordinarily succumb to the disease, there is no necropsy material available for study. Furthermore, since the osseous lesions are benign, no amputated extremities have been described except in the case reported by Telford,[7] that of a woman 34 years old who requested amputation of a long-deformed lower extremity which showed 5 inches (12.5 cm.) of shortening.

This patient sustained a fracture of the femur at the age of 14, after which swelling of the tibia and progressive shortening of the limb were noted. Examination of the surgical specimen showed the entire femur

7. Telford, E. D.: A Case of Osteitis Fibrosa, with Formation of Hyaline Cartilage, Brit. J. Surg. **18**:409, 1931.

Fig. 16.—*A*, roentgenogram of the lower end of the right femur in case 4. *B*, roentgenogram of the right tibia in case 4.

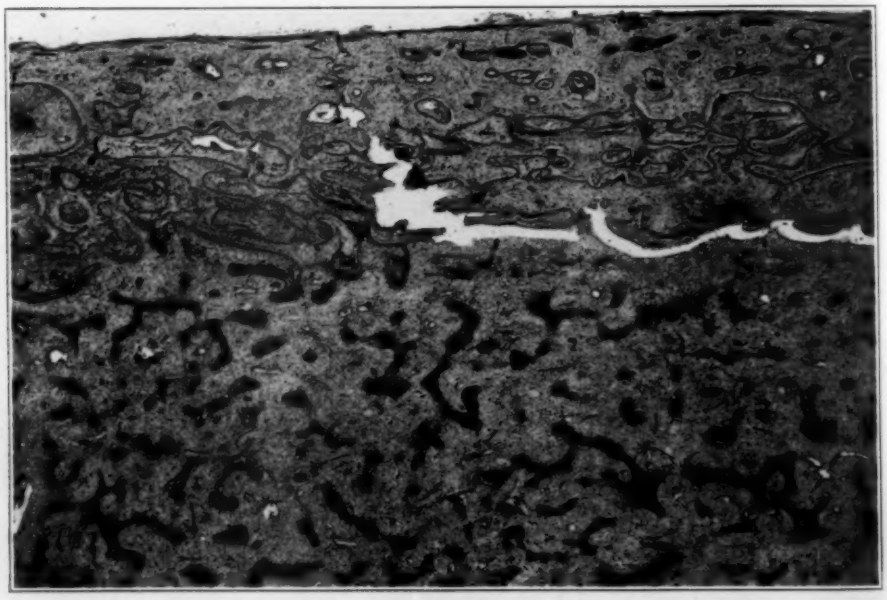

Fig. 17.—Biopsy specimen of the right tibia in case 4, longitudinal section, illustrating the absence of deposition of periosteal bone, the thinning of the cortex with erosion of the endosteal surface by fibrous tissue, which also replaces the substantia spongiosa and fills the medullary cavity and the deposition of trabeculae of primitive bone in the fibrous tissue (× 15).

and tibia, particularly the femur, to be much expanded, deformed and shortened. The cortical bone was reduced to a thin shell, and the medullary cavity was almost completely filled in by a white, fibrous, tough, gritty tissue, containing a few focal areas of hyaline cartilage. Histologic sections, which Professor Telford kindly permitted me to examine, closely resembled those prepared from my own material and indicated that the condition was an advanced stage of polyostotic fibrous dysplasia, limited, roentgenographically at least, to the left lower extremity.

The paucity of satisfactory pathologic material makes it all the more necessary to reconstruct a comprehensive picture of the osseous lesions from the biopsy specimens that are available. My concept of the pathologic nature of the disease is based on a study of 4 adequate specimens taken for biopsy from patients in this hospital, substantiated by gross and microscopic examination of material from 4 other patients submitted to Dr. Henry L. Jaffe from other hospitals. In each instance in which biopsy was performed for diagnosis in the cases in this hospital, both the surgeon and the pathologist concurred in the observation that the cortex was much thinned and that the medullary cavity was filled by fibrous tissue (fig. 1). The latter was described as grayish white or simply whitish; it had a peculiar consistency, variously described as spongy, elastic or rubbery, and was noted to be gritty (because of spicules of calcified new bone). Histologic examination of this fibrous tissue showed definite characteristic features, to be discussed presently. No cysts were encountered in any of the specimens, either on gross or on microscopic examination.

Thinning of the cortical bone, which may eventually be reduced to a thin expanded shell, is due in part to resorption but largely also to erosion of the endosteal surface by the proliferating fibrous tissue. There is, however, no gross or microscopic evidence of periosteal deposition of new bone, except in connection with callus at the site of spontaneous infractions of the cortex. The substantia spongiosa and also the marrow of affected bones are replaced by fibrous tissue. The latter (fig. 14) is composed of spindle cells with oval, pale-staining nuclei and indistinct cytoplasmic outline. The basic connective tissue may in some areas be fairly cellular, while in other areas, on the contrary, there may be a fibroblastic differentiation with a formation of bundles of mature connective tissue, rich in collagen (fig. 13). Special stains for detecting lipoid gave negative results. The stroma may be infiltrated by a few small lymphocytes and mononuclear cells.

Dispersed within the fibrous tissue without following any definite pattern, there are small trabeculae of primitive, poorly calcified new bone of variable size and contour. They are apparently developed by osseous metaplasia, without the mediacy of osteoblasts. In the relatively

avascular fibrous areas the trabeculae tend to be sparse in distribution; in the more vascular areas there is usually more extensive deposition of osteoid material and fiber bone (fig. 9). Some of the trabeculae show Howship's lacunas lodging osteoclasts, but on the whole there is comparatively little evidence of osteoclastic resorption. Deposition of calcium within the primitive new bone is variable in extent and distribution. Generally speaking, the bone is calcified poorly and in an imperfect, spotty fashion.

The basic fibrous tissue, on the whole, is relatively avascular, showing only occasional thin-walled vascular spaces. In some areas, however, it may be permeated by a moderate number of capillaries and even occasional small arterioles. Many of the blood vessels are in close proximity to the trabeculae of the bone. The latter are frequently surrounded by slender spaces which because of their endothelium-like lining suggest vascular spaces. Some capillaries appear empty, while others are congested and show small perivascular extravasations of blood. The presence of granules of hemosiderin within phagocytes indicates previous capillary hemorrhages which have been resorbed. In their vicinity one may also encounter small nests of giant cells, resembling osteoclasts. These multinuclear cells are basophilic and may have as many as ten or more nuclei; they are apparently formed by direct transformation and coalescence of the cells of the stroma. While some of the giant cells are distributed at the periphery of osseous trabeculae, thus suggesting an osteoclastic function, most of them are situated in the soft fibrous tissue. Sporadic islands of hyaline cartilage (fig. 15) may also occur within the fibrous tissue, although this is not constantly observed. The cartilage tends to become calcified, especially at its periphery. The significance of this feature will be discussed presently.

PATHOGENESIS

The characteristic pathologic feature of polyostotic fibrous dysplasia appears to be a disturbed function or development of the bone-forming mesenchyme, which results in replacement of the substantia spongiosa and filling of the medullary cavity of affected bones by fibrous tissue in which trabeculae of poorly calcified primitive new bone are developed by osseous metaplasia. The seemingly complex histologic picture becomes much easier to interpret if one predicates the multipotent character of this undifferentiated fibrous tissue, which normally gives rise to the substantia spongiosa and to the myeloid substances or fatty marrow but under pathologic conditions may develop in several anomalous ways. By osseous metaplasia, it gives rise to osteoid material and primitive fiber bone. By cartilaginous metaplasia, it gives rise to sporadic, isolated islands of hyaline cartilage, which tend to become calcified. By fibro-

blastic differentiation, it gives rise to mature collagenous connective tissue. Finally by coalescence of its nuclei, it may give rise to multinuclear cells, indistinguishable from osteoclasts. They apparently represent skeletal phagocytes and are of minor importance so far as pathogenesis is concerned.

The various potentialities of the basic connective tissue may be represented schematically as in figure 18.

The relative extent of the deposition of new bone and the formation of mature connective tissue is variable, depending apparently on the stage and vascularity of the lesion, the severity of the disease and indeterminate factors. Generally speaking, in poorly vascularized areas within the basic fibrous tissue much collagen and little bone are formed; in well vascularized areas deposition of bone proceeds more rapidly. Also, in severe forms with more extensive osseous involvement the basic fibrous tissue shows a tendency to more pronounced osteogenic differentiation. The histologic picture in any given instance may

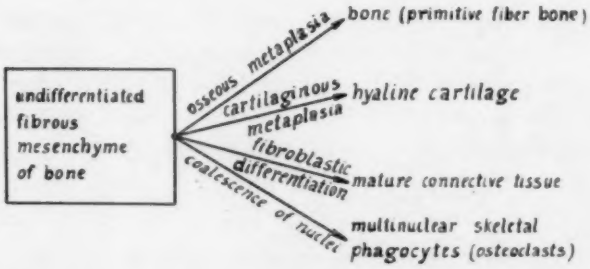

Fig. 18.—Schema showing the various potentialities of basic connective tissue.

run the whole gamut from extensive areas of mature connective tissue with sparse bony trabeculae (fig. 13), on the one hand, to areas composed largely of trabeculae of osteoid material and fiber bone, on the other (fig. 9). The amount of hyaline cartilage deposited is relatively small and indeed may not be detected in limited specimens taken for biopsy. Its development even in small quantity is nevertheless of considerable theoretic interest, in that it suggests a possible relation to another anomaly of skeletal development, skeletal enchondromatosis.

There is no histologic evidence to suggest that the condition is truly neoplastic. Even in the more cellular areas, there are no mitoses and no cellular atypism is noted. Clinically, also, it may be pointed out, no instance of malignant degeneration has been recorded. Nor is there any evidence that the condition is inflammatory in nature. While there may be small foci of lymphocytic infiltration within the fibrous tissue, they are not constantly observed and even when noted are insignificant. Whatever stimulates the continued perverted activity of the undiffer-

entiated fibrous bone-forming mesenchyme, or initiates the disorder, remains of course a matter of conjecture. The clinical histories consistently describing symptoms dating back to early childhood strongly suggest a congenital basis for this curious anomaly of skeletal development. To my knowledge no instance of hereditary transmission of the disease has been recorded.

DIFFERENTIAL DIAGNOSIS

The common erroneous clinical diagnoses have for the most part already been indicated, the most frequent being that of hyperparathyroidism. Others include Recklinghausen's disease, unilateral Recklinghausen's disease, osteitis fibrosa cystica and "cystic disease." If only a single bone is examined roentgenographically, a mistaken diagnosis of giant cell tumor or of enchondroma may be made. In an occasional case unilateral involvement may even be mistaken for dyschondroplasia or Ollier's disease (skeletal enchondromatosis with a tendency to attack one side of the body). Traumatic coxa vara and juvenile osteomalacia also are diagnoses that may sometimes be made before further investigation reveals the real nature of the lesion. Paget's disease and malignant disease of the bone are two other possibilities which may sometimes be considered, but which can easily be dismissed after roentgenographic and pathologic examination.

Hyperparathyroidism (Osteitis Fibrosa Cystica, Recklinghausen's Disease of the Bone).—Hunter and Turnbull [8] (1931), who described in connection with their discussion of hyperparathyroidism 3 instances of the disease under the title "osteitis fibrosa in multiple foci," realized that the condition was not Recklinghausen's disease and warned against unnecessary exploration. Jaffe,[9] likewise, considering the condition in connection with the differential diagnosis of hyperparathyroidism, emphasized that it has frequently been misinterpreted as Recklinghausen's or Paget's disease. He also recognized that it constitutes a still unidentified entity, having a possible congenital basis. Nevertheless, even at present, polyostotic fibrous dysplasia is frequently confused with hyperparathyroidism. This difficulty arises principally from the fact that the osseous lesions in the two diseases may roentgenographically have a striking resemblance, which is readily misleading to those who are as yet unfamiliar with the clinical picture of polyostotic fibrous dysplasia. In at least 6 instances, as reported in the literature, patients

8. Hunter, D., and Turnbull, H. M.: Hyperparathyroidism: Generalized Osteitis Fibrosa, Brit. J. Surg. **19**:203, 1931.

9. Jaffe, H. L.: Hyperparathyroidism (Recklinghausen's Disease of Bone), Arch. Path. **16**:63 (July); 236 (Aug.) 1933.

were subjected to exploration for parathyroid adenoma because of the mistaken clinical diagnosis of hyperparathyroidism. In each instance the parathyroid glands were found to be normal in size and appearance. Two additional patients were needlessly subjected to roentgen irradiation of the neck under the same erroneous premise.

Actually, however, in cases of well developed polyostotic fibrous dysplasia, the diagnosis is evident if one keeps the possibility in mind. The clinical history and roentgenographic observations, as previously indicated, are usually rather characteristic. Other differential features which help to rule out hyperparathyroidism are the high incidence of fibrous dysplasia in childhood and adolescence, its predominantly unilateral distribution and the fact that the unaffected bones appear normal roentgenographically, without evidence of porosis. Furthermore, the calcium in the serum in many cases of fibrous dysplasia is within the normal range. Moreover, studies in the balance of calcium, as reported by Albright and associates,[4] show no appreciable loss of calcium by excretion.

In some few equivocal cases, in which the osseous lesions are not roentgenographically definitive and in which the amounts of calcium and phosphatase in the serum are somewhat elevated (as they may be in polyostotic fibrous dysplasia), it would seem wise to excise a bit of one of the affected bones for biopsy before attempting exploration of the neck for parathyroid adenoma. With reference to the concept of unilateral Recklinghausen's disease proposed by some authors (Goldhamer [2] and Borak and Doll [3]) to explain instances of polyostotic fibrous dysplasia, one need simply reiterate that the disease is in fact neither exclusively unilateral nor due to hyperparathyroidism. Moreover, even the theory is unsound, since it is manifestly illogical to expect that a disease resulting from hyperfunction of endocrine glands would affect the bones of only one side of the body.

Skeletal Enchondromatosis (Dyschondroplasia or Ollier's Disease).—One of the less frequent errors in clinical diagnosis is to confuse polyostotic fibrous dysplasia with skeletal enchondromatosis. This arises from the fact that in both conditions the osseous involvement may sometimes be predominantly unilateral. However, the clinical and roentgenographic picture otherwise readily suffices to distinguish the two diseases. Other considerations which help to rule out skeletal enchondromatosis are the onset of symptoms in childhood or adolescence rather than early infancy, the absence of macroscopic enchondromas in the metacarpal, metatarsal and phalangeal bones and the less pronounced shortening of affected extremities in polyostotic fibrous dysplasia. Furthermore, a biopsy will definitely establish the diagnosis. While occasional small islands of hyaline cartilage may be found in polyostotic

fibrous dysplasia, the basic histologic picture is one of fibrous tissue in which small trabeculae of immature fiber bone are deposited.

Hand-Schüller-Christian Disease.—Young subjects are particularly susceptible also to Hand-Schüller-Christian disease, and it too may cause punched-out rarefied lesions, visible roentgenographically in the skull and other bones. Even pathologic fracture of the femur may be simulated. The resemblance is superficial, however, so that xanthomatosis of the bones due to cholesterol can be ruled out usually on clinical grounds, because of the absence of the Christian syndrome, and if necessary by biopsy.

POSSIBLE RELATION TO LOCALIZED LESIONS APPARENTLY AFFECTING ONE BONE

The relation of localized fibrous osseous lesions apparently affecting only one bone to polyostotic fibrous dysplasia is a problem that cannot be definitely decided at present. Freund and Meffert [5] have described two such localized lesions apparently limited to the femur, which roentgenographically and also on biopsy closely resemble the polyostotic form of the disease. Recently I examined sections, sent to Dr. Jaffe, of a biopsy specimen obtained from a girl, aged 14, who presented a solid expanded lesion of the maxilla of the jaw. The condition was diagnosed clinically as central osteoma. The histologic appearance of the lesion, however, was indistinguishable from that seen in polyostotic fibrous dysplasia. Although there were no symptoms referable to other bones, roentgen examination of the skeleton, especially of the long bones, was recommended. Several similar cases have been described by Phemister and Grimson [10] as instances of fibrous osteoma of the jaw.

I have seen also a patient with a localized, expanded, rarefied lesion of the clavicle, diagnosed clinically as giant cell tumor or enchondroma. Biopsy showed the affected portion of the clavicle to be filled with whitish fibrous tissue such as is seen in polyostotic fibrous dysplasia. Roentgen examination of the other bones disclosed only a few suggestive lesions in the ribs.

Localized fibrous lesions of the type described may be regarded as representing a limited form of polyostotic fibrous dysplasia. Since the evolution of the disease is relatively slow, it would not be at all surprising to observe that other osseous defects subsequently develop in these patients. Another possibility is that there *are* multiple osseous lesions in these instances but that some of them are not sufficiently well developed to be visualized roentgenographically.

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10. Phemister, D. B., and Grimson, K. S.: Fibrous Osteoma of the Jaws, Ann. Surg. **105**:564, 1937.

PROGNOSIS AND TREATMENT

So far as is known, polyostotic fibrous dysplasia is a chronic, slowly developing disease, which becomes manifest generally in childhood and persists throughout adult life. Indeed, I have observed two representative cases, with diagnosis proved by biopsy, in patients in the fifth decade of life. The condition is not self limited in the sense that affected bones are ever restored to their normal structure. Study of the available material for biopsy has indicated, however, that in older patients there may be demonstrably less evidence of activity of the lesions and that a sort of static equilibrium may eventually be established. There is of course no specific medical therapy available.

Elective surgical procedures employing curettage and bone grafts for the purpose of strengthening extensively involved, attenuated and expanded bones do not seem altogether warranted, since the same fibrous tissue fills in again. Furthermore, operative wounds may heal relatively slowly, and pain may be aggravated by surgical intervention rather than alleviated. In one of my patients, there were delayed healing and persistent pain two years after partial ostectomy of the tibia. In other instances, six months to a year was required for solid bony union. Surgical intervention, then, should be limited to the treatment of spontaneous fractures and to osteotomy in selected cases, for the correction of severe bowing of the long bones. Because of the pronounced tendency to spontaneous fracture in extensively involved bones, principally in the neck of the femur and occasionally in the humerus, patients should be cautioned to avoid minor traumas and to refrain from unduly strenuous exercise or other activity which might induce a fracture. It would appear worth while to try roentgen irradiation in small or moderate dosage, to ascertain empirically whether it has any beneficial effect. However, it is important to bear in mind that large doses may lead to radiation necrosis of the thinned cortical bone, with resulting pathologic fracture. I know of such an instance.

SUMMARY

Polyostotic fibrous dysplasia, as I conceive it, is a skeletal developmental anomaly, usually affecting several or many bones, with predominantly unilateral involvement. Lesions on the opposite side of the body do occur, however. The disease is by no means uncommon and is observed most often in children and young adults. The available data indicate, further, a definite tendency to occur in women. The cause is unknown; however, symptoms consistently dating back to early childhood suggest that the disorder is congenital, but evolves slowly. Unlike some of the other diseases resulting from anomalous skeletal develop-

ment, e. g., multiple exostosis and achondroplasia, no familial incidence or hereditary tendency has thus far been noted.

The long bones of the lower extremity (femur and tibia) are most frequently affected, but those of the upper extremity (humerus and radius) and also the skull, ribs, pelvis, phalanges and other bones may be involved. Epiphysioid bones (carpals and tarsals) and bones preformed in membrane as well as in cartilage may be affected. The epiphyses of affected bones as well as their metaphysial and diaphysial regions may be involved. The condition runs a slow, progressive clinical course over a period of many years, even decades, with a tendency to spontaneous fracture and deformity of affected bones. The characteristic pathologic feature appears to be a disturbed function or development of the bone-forming mesenchyme, resulting in the filling of the medullary cavity of affected bones by fibrous tissue, in which spicules of poorly calcified primitive fiber bone are developed by osseous metaplasia. Some areas, however, may show a predominantly collagenous differentiation with comparatively little deposition of new bone. Furthermore, small islands of hyaline cartilage (remote from the epiphysial plate) may also be formed by chondrogenic metaplasia.

The aforementioned changes result in prominent widening of affected bones, marked thinning of their cortices and replacement of the cancellous bone and bone marrow by a fibrous, whitish, gritty, solid tissue. The bones on roentgen examination cast a shadow of diminished density, suggesting a rarefied or porotic trabeculated lesion, replacing bone and usually interpreted as indicating cystic disease. Thus, the condition with which polyostotic fibrous dysplasia is most often confused is ostitis fibrosa cystica (Recklinghausen's disease of bone). The not infrequent observation of increased values for calcium and phosphatase in the serum in cases of fibrous dysplasia affords another pitfall, suggesting the erroneous diagnosis of hyperparathyroidism. Indeed many patients suffering from polyostotic fibrous dysplasia are subjected to exploration of the neck for parathyroid adenoma only to have the surgeon find normal parathyroid glands. The differentiation from other skeletal diseases has been discussed.